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<b>Physician's Handbook, 11th edition</b> H. A. Krupp, H. I. Sweet, E. Brown, and C. D. Armstrong	\$2.50 1946
<b>Handbook of Medical Treatment, 2d edition</b> M. J. Carter, B. Morgan, and H. D. Brunsford	\$3.00 <i>revised October, 1934</i>
<b>Handbook of Obstetrics and Diagnostic Gynecology</b> <i>2nd edition</i> L. Doyle	\$2.25 1943
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# HANDBOOK of MEDICAL TREATMENT

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## Chapter 1

# GENERAL ASPECTS OF MEDICAL TREATMENT

Successful medical treatment includes consideration of all the resources available for achieving normal functioning of the physical and mental status of the patient during the course of his illness. The physician must consider the following general and specific measures in formulating a therapeutic regimen:

1. Activity status and bed position	Page 1 and 2
2. Environment (including home management)	Page 2
3. Clinical observations	Page 3
4. Laboratory studies	Page 3
5. Fluids	Page 3 and 10
6. Symptomatic and supportive measures	Page 30
7. Diet	Page 44
8. Specific measures	See Specific Disease

## ACTIVITY STATUS

Bed rest has long been and will continue to be a basic treatment modality for many illnesses. It is, however, not without its disadvantages or even its dangers.

The degree of activity permitted a patient should be based upon a careful consideration of the patient's physiological needs for activity or rest. In general, activity should be less than that which interferes with healing processes or induces respiratory distress or other undesirable symptoms (e.g., excessive fatigue). On the other hand, activity should not be limited to such a extent that disadvantageous physiological or psychological results ensue.

### Type of Activity Status

- A. Ambulatory. For ill patients unable to perform specific coordinated activities (see below).
- B. Bed Rest With Bathroom Privilege. For those patients in whom full mobility is temporarily contraindicated.
- C. Bed Rest Without Bathroom Privilege. For most patients with:
  1. Continued disease
  2. A state of moderate to severe degree
  3. Moderate to marked dyspnea from most causes
  4. Local infections and inflammation (e.g., cellulitis, phlebitis, etc.) especially of the lower extremities
- D. Complete Bed Rest. The patient is required to remain in physical inactivity and relief from emotional stress or excitement. They must have assistance in eating, changing body position, and using the bathroom. Complete bed rest is recommended for patients with:



# **BED POSITIONS** (and Indications)



## **SHOCK**

Vasomotor collapse (primary and secondary shock)



## **SEMI FOWLER**

Dyspnea from any cause



## **FOWLER**

Marked dyspnea from any cause

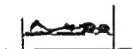
## **HEAD OF BED ELEVATED**

In bed but a partial prone position



## **CARDIAC BED**

Marked dyspnea from any cause (more satisfactory than Fowler)



## **ARTIFICIAL PRONE**

Artificial respiration  
Decubitus ulcers of back  
Vomiting or obstructed respiration in comatose patients



## **LEGS ELEVATED**

Infection inflammation phlebitis or edema of the lower extremities



## **GENU PECTORAL (knee chest)**

Dyspnea in children  
Rectal and sigmoidoscopic examination



## **TRENDELENBURG**

Following pelvic operations

patients cold sensitive patients and patients in vasomotor collapse

### Room Humidity

- A Relative humidity of 60% is considered ideal with an accepted range of 35-70%. In general, higher humidities are tolerated better with lower room temperatures and vice versa
- B Higher humidities may be of value in the treatment of patients with asthma

### Ventilation

Circulation of air may be as essential to comfort as proper warmth and humidity especially in certain respiratory and cardiac illnesses. Avoid drafts

### Social Contacts

- A Restriction of Social Activities This includes curtailment of visitors telephone radio and letter writing when indicated. These limitations should seldom if ever be imposed over a long period of time. They apply principally to:
  - 1 Seriously ill patients
  - 2 Patients with contagious diseases
  - 3 Certain disturbed, psychotic or delirious patients and acutely hysterical patients (However relatives and other familiar figures may lessen confinement and relieve anxiety in some cases)
  - 4 Markedly dyspneic patients
- B Encouragement of Social Activities
  - 1 Most patients not in the above categories
  - 2 Chronically ill patients
  - 3 Certain depressed emotional states

### Special Consideration

- A Elimination of noise
- B Darkening of room for patients with photophobia or with tetanus. Not advised for delirious patients
- C Elimination of dust in cases with respiratory diseases or dust allergies
- D Elimination of allergenic materials in pillows bed linen curtains room furnishings etc for allergic patients

### Factors in Recommendation of Home Management for Bed Patients

- A General
  - 1 Hospital facilities in community
  - 2 Financial status of patient in general, if patient able to afford it hospital care is preferable for bed patients
  - 3 Proximity of patient's home to physician's office
  - 4 Character of disease ( dangers to patient attendants members of the family and community )
- B Home Factors
  - 1 Home facilities (space heat electricity furniture etc )
  - 2 Intelligence and cooperativeness of patient and family
  - 3 Nursing care Availability of desirable attendants (may be required night and day)
  - 4 Duration of illness (consider expense of prolonged hospitalization)

General Rules for Home Management of CasesA Preparation of the Room

- 1 Move patient to a table room considering
  - a Ventilation
  - b Temperature
  - c Electrical fixtures
  - d Bathroom facilities
- 2 Rent hospital bed or use plain narrow bed raised on blocks to convenient height for attendant
- 3 Remove all unnecessary furniture and accessories from room especially in isolation cases
- 4 Use simple bed linen and blankets

B Attending

- 1 Obtain services of trained or practical nurse for complicated or serious contagious cases
- 2 Instruct attendant concerning patient's disease with specific objectives danger signals etc. If necessary dictated action in writing
- 3 Explain medical situation to responsible person emphasizing observance of following instructions
  - a Activity status of patient
  - b Proper bed care
  - c Proper diet and fluids
  - d Social contacts (visitors telephone correspondence)
  - e Regularity of treatment
  - f Informing physician of any unusual change in patient

Obtaining Home Nursing Service

The director of the local telephone company community health or health council provides leads to the official registration for full, part time or hourly professional and practical nursing services. These agencies may assist the physician in arranging for other home service as indicated.

Contagious Disease Management in the HomeA Consultation Requirements

- 1 Formulate in your mind with the isolation and quarantine requirements of the disease in question, as applicable to your community. Determine whether or not the disease is reportable
- 2 Emphasize the family's responsibility to the community

B Preparation for Isolation

- 1 State the important points of isolation technique as the case demands. Point out the signs of spread
- 2 Attempt to approximate hospital isolation technique
- 3 Utilize service of attendants who have either had the disease or who have been immunized against it. Either immunize or remove susceptibles from the home. If necessary obtain the services of a trained nurse

C Isolation Technique

- 1 Define and designate the isolated room or area of the house. The selected area is to be used by the patient and attendants and NO OTHERS. Children are never to enter this area. Instruct all patients to wash hands in soap and water carefully before entering and on leaving the room
- 2 Disposable paper masks and caps are now available quite cheaply or large handkerchiefs may be used as masks or

## 4 Clinical Observations

Temperatures and other vital signs should be taken in the room and should be recorded and removed in the center of the table. A list of the patient's (inside of glove and of an)

### CLINICAL OBSERVATIONS

Valid and reliable information of the patient as well as diagnostic also may be gathered from a carefully maintained clinical record. Such information provides additional data about the patient at the present time and prognosis of the disease. This helps in sure the general condition of the patient. The following brief clinical data are included for the conversion of the clinician in form of a table into a list of the patient's. The physician's task is considerably simplified if the patient is in a hospital where a nurse nursing the patient is a skilled nursing supervisor and careful observation of patient's activities, vital signs, medical history and other important data is possible and more effective management of the patient.

#### Body Temperature

A list of the patient's temperature and other vital signs

Area	Average Temp	Range of Temp
Rectal	99.6°F (37.5°C)	98.5-99.8°F (37.5-37.7°C)
Oral	98.6°F (37.0°C)	96.5-99.8°F (37.5-37.7°C)
Axillary	97.6°F (35.5°C)	96.5-98.0°F (36.5-37.4°C)

#### Body Temperature

##### 1 Types

- Remittent** Of days or weeks duration with alternating periods during which temperature is normal (e.g. bacterial infections or infectious malarial). Temperature should be taken at least once a day for a prolonged period (weeks to months) to determine the alternating febrile and afebrile periods.
- Intermittent** Temperature drops to normal or subnormal level once or more in 24 hours (e.g. septic fevers and early tuberculous). Temperature must be taken at least once a day to determine the variation within the day.
- Continuous** Temperature remains normal during 24-hour period (e.g. pneumonia, influenza). Temperature must be taken at least once a day or at times every 2-3 hours to determine the sustained character of the fever.

2 Causes Infectious diseases, certain drugs for immunoprotein responses, viral and bacterial diseases, disturbance of heat regulating center and neuroses.

- Normal temperature** May be due to profuse perspiration, hemorrhage, shock, decreased blood flow, and mental depression. A common cause of recorded subnormal temperature is insufficient time allowed for taking temperature. Subnormal temperatures may indicate failure in a seriously ill patient and demand





## 8 General Observations

- 1 General rule For every degree (F) of temperature rise the pulse rate usually rises 10 beats per minute i.e.  
98 60/min 99 70/min
- 2 Diseases in which pulse rate may be low in proportion to fever (relative bradycardia) Typhoid fever undulant fever brucella meningitis infectious mononucleosis
- 3 Diseases in which pulse rate is usually high in proportion to fever (relative tachycardia) Scarlet fever rheumatic fever diphtheria thyrotoxicosis subacute bacterial endocarditis tuberculosis terminal or unfavorable pneumonia (pre shock)

### B Respiration Temperature Relationships

- 1 General rule Respiratory rate roughly parallels temperature changes
- 2 Exceptions Intrathoracic or respiratory diseases (relative tachypnea, hyperpnea or dyspnea)

### C Pulse Blood Pressure Relationships

- 1 General rule The same factor causing an increase in cardiac rate usually causes an increase in blood pressure
- 2 Exceptions
  - a Relative tachycardia Same as for pathological causes of hypotension
  - b Relative bradycardia Renal disease benign and malignant hypertension, increased intracranial pressure

### D Mental Status Temperature Relationships

- 1 General rule Delirium in the company high fevers
- 2 Exceptions (when patients are more susceptible to febrile delirium i.e. with lower fevers) Emotional lability postoperative typhus fevers and the influence of certain drugs (e.g. barbiturates)

## Miscellaneous Observations and Precautions

The following clinical observations are also important in determining the general comfort and in following the clinical status of the patient

- A Fluid Intake and Output Consideration of the fluid balance of the patient should include the following
  - 1 Clinical evaluation of state of hydration
  - 2 Estimation of need for fluids
  - 3 Types of fluid administration(For details see Chapter 2 page 10)

- B Condition of the Skin Evidence of decubiti (bed sores) heat rash, hyperhidrosis drug rashes etc

- C Condition of the Mouth Lips and Nares Evidence of chafing ulceration soreness dehydration avitaminosis etc

Local conditions in the mouth permitting patients should brush their teeth or have their teeth brushed with some simple dentifrice at least once daily

Patients should be given the opportunity to rinse their mouths after each meal Plain tap water physiological saline solution, or Alkaline Aromatic Solution N.F. (diluted 2:1) are equally satisfactory

Care must be taken that the patient is in an adequate state of nutrition and hydration

- D Appetite Question patient regarding appetite and qualitative and quantitative food desires Check actual food intake by

## Laboratory and X Ray #

- examination of outgoing trays. Determine reasons for rejection of food. Avoid prologation of unpalatable or restricted diets for periods in excess of actual requirement (see section on Diet, page 44).
- E Elimination.** Bed patients are generally prone to constipation. This may be exaggerated by the illness itself, diet, or bed pans, diets, and rectal drugs. When knowledge of elimination is especially important, gross inspection of all stools passed by the patient may be necessary (see Constipation, page 334). Daily inquiry regarding elimination should be made of each patient.
- F A plan for Rejection of Medication.** Always inquire as to patient's plan of medication. The patient's objections may constitute a valid reason for modification or cessation of drug therapy. Side effects as to untoward reactions from medication also deserve careful attention.
- G Sleep and Rest.** The physician should inquire about amount of sleep or rest, vary considerably from known observations. Provide suitable environment for sleeping and resting by insuring a minimum of interruptions by professional attention. Sleep inducing drugs should be avoided (see Insomnia, page 33).
- H Monitor Reaction of Patient.** Observe patient's mood and behavior, reflectively in his mental depression, which is often associated with costly continuing serious or chronic illness.

## LABORATORY AND X RAY STUDIES

### Ordering Laboratory and X ray Studies

- The clinical and rational ordering and performance of laboratory, x-ray, diagnostic, and other special studies constitute an essential phase of the management of the patient. The blood count and analysis of serological test for syphilis and perhaps chest x-ray should be performed routinely on all hospital patients.
- A Special diagnostic studies may conflict for careful planning and integrating with the therapeutic program and must not conflict with the treatment scheduled.**
- B Improperly performed or unnecessary laboratory and x-ray studies, aside from the discomfort, expense and inconvenience they cause the patient, may prolong hospitalization.**
- C It must be remembered that certain laboratory studies may require dehydration (e.g., Adonis test or pyelograms) which may be clinically dangerous (e.g., precipitation of renal failure, etc.).**
- D Likewise, forced fluids (e.g., F&S P) may be contraindicated in the presence of nausea or severe congestive failure, etc.**
- E X-ray Fluorine and dye studies should precede all biologic contrast studies, and retrograde biliary (cholangioma) studies should precede upper gastrointestinal studies. Inverse reversal of this sequence of studies will cause a needless delay.**

## Chapter 2

# FLUID AND ELECTROLYTE THERAPY AND PARENTERAL FEEDING

## FLUID BALANCE

In clinical fluid therapy it is necessary that the problems of water and electrolyte metabolism be considered independently of each other. The electrolytes are intimately concerned with the maintenance of normal cellular metabolism, acid-base regulation and with water the maintenance of osmotic pressure in both the extracellular and intracellular fluid compartments. Water in excess of the quantity necessary for maintaining the tonicity of body fluids is required for normal bodily function. The administration of solutions of isotonic electrolyte to the patient cannot be considered as providing a balance with respect to the excretion of these same electrolytes. Water must likewise be excreted to keep the solution (urine) almost isotonic.

### Daily Obligatory Water Requirement is

A certain minimum amount of water (free from electrolytes) is necessary for normal bodily function. These obligatory water requirements are related to energy expenditure. They are given in the following table.

### AVERAGE DAILY WATER NEEDS FOR EXCRETION

Method of Excretion or Loss	Volume of Water Excreted per 100 Calories of Food	Volume of Water Excreted per Day
Imperceptible loss (lungs and skin)	44 cc	1100 cc
Sweat (imperceptible)	Varies with external temperature. May be high	0-300 (more at high temperature)
Urine	Varies with amount of waste products to be excreted (see chart page 12)	Average minimum 1000 cc (if upgr more than 1,000)
Needs for excretion other than urine		1200-1500
Total (including urine)		2200-2500

Based on 2500 Calories food intake

For gross clinical estimation the imperceptible loss of water may be calculated as 10 cc /K (5 cc /lb ) body weight per day



Specific Gravity	Gm Solids per Liter	Urine Vol / 33 Gm Solids (Avg Sp Gr)	Urine Vol / 30 Gm Solids (Avg Sp Gr)
1.035 - 1.030	81.79	400	800
1.030 - 1.025	79.87	475	950
1.025 - 1.020	87.35	570	1140
1.020 - 1.015	55.43	715	1430
1.015 - 1.010	43.31	950	1900
1.010 - 1.005	31.19	1400	2800

Any dilution and marked change in the patient's hydration status as compared to the latter portion of the collection period will fail to be reflected in the specific gravity of the total 4-hour specimen.

## ROUTES AND TYPES OF WATER ADMINISTRATION

### Amount and Availability of Water for Metabolism

- A Oral Liquid route. In calculating fluid intake remember that most foods contain water and that about 12% of all calories metabolized form water (of oxidation).
- B Parenteral. When considering water balance note that:
  1. The water in glucose solutions is all available for metabolic and excretory purposes.
  2. Solutions containing electrolytes (e.g., normal saline) yield little water for metabolic purposes for the electrolytes require water for excretion.
  3. Protein hydrolyzates. 1 Gm of protein equals about 1/3 Gm of urea for excretion in urine. Therefore depending on urinary specific gravity some water may be available from 5% protein hydrolyzates if the electrolyte content is low (see page 9).
- C Rectal. Rarely employed. Water or 2% glucose in water may be administered by slow drip if no large bowel disease is present. This is all available for metabolic purposes.

### Types of Water Regimens

- A Liberal (or dilute). Indicated for the average patient in a normal physiologic state when dehydration is not present. 1800-3000 cc of total fluids daily are adequate except for increased loss by sweating.
- B Restrictive. May be indicated when factors are working which tend to cause accumulation of body fluids. Restriction of oral fluids may be indicated when for any reason it is undesirable to introduce more than small quantities of fluid into the GI tract.
  1. Anuria or oliguria (e.g., lower nephron syndrome - see page 303).
  2. In conjunction with ketogenic diet.
  3. Preparation for certain laboratory studies (Addis test, pyelography).
  4. Restriction of oral fluids. Conditions interfering with passage of fluids through the gastrointestinal tract e.g., swallowing disorders, persistent nausea and vomiting, gastric dilatation and intestinal obstruction. When these conditions are present it is necessary to replace fluids by the parenteral route (see next page).

**Indications (Forced):** May be indicated when fluids are working but not enough to replace body fluids or when it is desired to hasten excretion of toxins or in tacholite. The addition of fluids must frequently be given by parenteral routes.

- 1 High atmospheric temperature
- 2 High body temperature (fever)
- 3 Diabetes (e.g. diabetes insipidus) or renal insufficiency with polyuria
- 4 Exudations from inflamed surfaces (especially severe burns)
- 5 Diarrhea
- 6 Vomiting
- 7 Draining fistula
- 8 Exogenous or endogenous poison. Heavy metal poisoning, renal failure (except when oliguria or anuria is of renal origin), diabetic coma, etc.

## ELECTROLYTE AND ACID BASE BALANCE

Normal concentration of both intracellular and extracellular electrolytes is necessary for life. Although the intracellular and extracellular electrolytes are at approximately the same osmotic pressure, the individual ions are different. The electrolytes present in the body are grouped into positive ions (cations) and negative ions (anions). The cations are concerned with the functions of the electrolytes. The anions apparently retard direct pharmacologic action but are intimately involved with ionic equilibrium. The following table gives both intracellular and extracellular electrolytes grouped as cations and anions.

VALUES OF EXTRACELLULAR AND INTRACELLULAR ELECTROLYTES

Ion	Extracellular			Intracellular	
	mEq/liter	mg/100 cc	mg/100 cc	mEq/liter	mg/100
	g	mg		g	
<b>Positive</b>					
Na	142	135-147	310-340	13	30
K <sup>+</sup>	5	4.0-5.0	18-22	140	350
Ca <sup>++</sup>	5	4.5-5.5	9-11	0	0
Mg <sup>++</sup>	3	3.5-3.0	1.8-2.8	45	54
<b>Total</b>	<b>155</b>			<b>198</b>	
<b>Negative</b>					
HCO <sub>3</sub>	27	25-30	36-45	10	22
Cl	103	100-110	350-390	3	10
HPO <sub>4</sub>	2	1.8-2.3	3-4	100	200
SO <sub>4</sub>	1		4-8	20	86
Org. Ac	8			0	0
Pot. in	16			65	
<b>Total</b>	<b>155</b>			<b>198</b>	

These are approximate values in muscle

\*Volume %

From the previous page it is a correct statement that the electrolyte content of the intracellular and extracellular compartments are entirely different. Whereas sodium chloride is the main component of extracellular fluid but potassium has possibly a part in it. Potassium is the main component of intracellular fluid. This is of foremost importance in consideration of the electrolyte balance (see page 21) especially as the extracellular components are available for clinical measurement.

Since little is known regarding the functions and uses of many of the intracellular electrolyte concentrations, diagnosis of the electrolyte must be concerned primarily with the extracellular fluid only. As a knowledge of the extracellular electrolyte concentration and the normal clinical laboratory procedures can be drawn from the laboratory of clinical investigation.

### VOLUME AND ELECTROLYTE CONTENT OF GASTROINTESTINAL SECRETIONS AND SWEAT\*

Source	Average 24 hr Volume	Electrolytes in mEq/L			
		Na	K	Cl	HCO <sub>3</sub>
Extracellular fluid		145	5	111	8
Gastric Juice					
Containing A	2500	10-110	1-32	8-155	0
Achlorhydria		80-120	1-30	100	30
Saliva	500	130-160	2-12	90-120	38
Pancreatic Juice	700	110-130	2-8	50-95	110
Small Bowel Secretion	100-8000	80-130	2-8	40-135	30
Ileostomy					
Recent	100-4000	100-130	5-30	90-140	80
Adapted	100-500	80	3	70	15-30
Cecostomy	100-3000	30	8	40	15
Urine					
(Formed)	100	< 10	< 10	< 15	< 15
Sweat	500-10,000	0-100	0-5	0-100	0

Minor alterations in ion concentration occur in interstitial fluid in response to physical laws governing the production of an ultrafiltrate of plasma.

Modified from Lockwood and Randall: *Bull. N. Y. Acad. Med.* 25: 228, 1949.

Reprinted from Krupp, Sweet, Jewetz, and Armstrong: *Physician's Handbook*, 8th ed. Lange Medical Publications.

## FUNCTIONS OF THE ELECTROLYTES

The electrolytes of the extracellular fluid serve three principal functions.

### REGULATION OF OSMOTIC PRESSURE AND WATER BALANCE

The osmotic pressure of both the extracellular and intracellular components of the body are at all times equal. In health the osmotic pressure is equal to about 310 milliosmoles per liter. The total body water is equal to about 45-65% of the body weight, with an average of 55% for males and 47% for females. The values are lower in obese and higher in lean muscular individuals. About 15-17% of the body weight is in the extracellular fluid compartment and about 43% of this is in the vascular compartment.

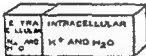
Because the ionic composition of the extracellular and intracellular fluids is entirely different and this difference is regulated by the cellular osmotic system which remove unwanted electrolytes (eg sodium) extrinsically as it diffuses into the cell with water.

Water balance is a function of osmotic pressure. The principal means of maintaining osmotic equilibrium whenever there are alterations in electrolyte or water concentration in the body. The significant alterations that now become clinically are illustrated below. The diagrams are oversimplified and fail to show the electrolyte shifts that occur in pathological conditions. Clinicalists generally discuss complex problems of this rather than simple entities.

### FLUID COMPARTMENTS

#### Normal

This figure represents the body water with the normal extracellular and intracellular fluid and electrolyte concentration.

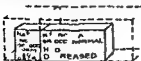


NORMAL

ECF (EXTRACELLULAR FLUID)  $K$  (CONSTANT FOR A)  
ICF (INTRACELLULAR FLUID) (GIVEN INDIVIDUAL)

#### Simple Dehydration Without Salt Loss

In this condition the electrolyte is concentrated the extracellular and intracellular fluid ratio the same as normal.



SIMPLE  
DEHYDRATION

$$\frac{\text{ECF}}{\text{ICF}} = K$$



## 10 Function of Electrolytes

- A Common Clinical Conditions Lack of water gastric vomiting  
B Diagnostic Points Marked thirst (probably a symptom of intracellular dehydration) poor tissue turgor high urine specific gravity high hematocrit all extracellular electrolytes may be elevated but proportions normal  
C Treatment Administer fluid without electrolytes Water orally 3-10% glucose in water I.V.

### Simple Overhydration

In this condition the electrolytes are diluted the extracellular/intracellular fluid ratio is the same as for normal



- A Common Clinical Conditions Excessive fluid intake without salt Excessive electrolyte free fluid administration to a patient with oliguria or anuria  
B Diagnostic Points Edema low urine specific gravity if patient urinating low hematocrit all extracellular electrolytes reduced, but proportions normal Convulsions if extreme  
C Treatment Usually withholding of fluid and electrolytes

### Excessive Sodium Retention (or Rarely Intracellular K<sup>+</sup> Loss)

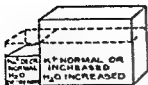
This leads to excess fluid in the extracellular compartment with depletion (dehydration) of the intracellular fluid



- A Common Clinical Conditions Cardiac failure liver failure with ascites renal disease with failure to excrete sodium excessive sodium intake administration of some steroid hormones or ACTH  
B Diagnostic Points Edema patient may be thirsty low hematocrit elevated blood pressure extracellular sodium may be normal or elevated urine specific gravity usually low  
C Treatment Restriction of dietary sodium or administration of agents to induce sodium loss by kidney (e.g. digitalis mercurials Diamox® etc) Water orally 3-10% glucose in water I.V. (to provide electrolyte free water)

### Excessive Na<sup>+</sup> Loss (Low Sodium Syndrome)

This leads to diminished extracellular fluid and an increase of intracellular fluid



## SODIUM LOSS

$$\frac{ECF}{ICF} < K$$

- A Common Clinical Condition Low sodium intake & excessive use of mercury diuretics excess fluid intake without sodium after prolonged sweating. Add on a disease sodium binding & in therapy sodium loss with gastrointestinal fluid loss &
- B Diagnostic Points Low blood pressure muscle cramps low urine volume because of thirst
- C Treatment Adrenal stimulation of sodium salts in once a day doses if still rather tonic (see page 22)

### MAINTENANCE OF NORMAL NEUROMUSCULAR FUNCTIONS (Neuromuscular Irritability)

The electrolyte content of the body is kept remarkably constant chiefly by the kidney's selective ability to excrete or conserve individual ions. The level and balance of the various positive ions (cations) is important in maintaining normal neuromuscular irritability and they vary little in health. Wide variations are incompatible with life & symptoms arise when a serious deficiency occurs.

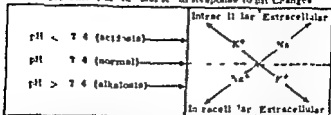
#### Interrelationship of Electrolyte and Neuromuscular Function

The interrelationship between electrolyte concentration and neuromuscular function is not clear. Many of the signs and symptoms of deficiency and excesses of ions may be due to alterations in intracellular contents which may or may not be detectable by the measurement of extracellular electrolytes and fluid alone.

- A Variation in Effect of Hormones An excellent example of this is found in the effect of two hormones upon serum potassium levels namely desoxytocorticosterone and testosterone. Both of these hormones can lower the serum potassium markedly. However symptoms of potassium deficiency never develop with testosterone. It is believed that this is because the hormone causes an intracellular movement of potassium and retention of potassium by the body. Desoxycorticosterone however causes marked urinary loss of potassium with intra and extracellular depletion and may lead to symptoms of potassium deficiency.
- B Effect of pH on Electrolyte The pH of the blood is important in determining the intracellular distribution of ions. This is most noticeable in the case of sodium and potassium. In a blood sodium is transferred from the extracellular to the intracellular sodium buffer system and is replaced intracellularly by potassium. (In addition intracellular potassium is lost to the body in acidosis.) In alkalosis the reverse occurs, sodium moves intracellularly while potassium leaves the cells. The changes are illustrated on the following page.

# III Neuromuscular Irritability

## Movement of $\text{Na}$ and $\text{K}$ in Response to pH Changes



## SYMPTOMS OF EXCESS AND DEFICIENCY OF THE POSITIVE IONS

Positive Ion (Cation)	Symptoms of Excess and Clinical Causes	Symptoms of Deficiency and Clinical Causes
<b>Sodium</b> $\text{Na}$  (Normal 142 mEq/L)	<i>Edema</i> Cardiac failure Cushing's syndrome Excessive sodium (Administration of some steroid hormones or ACTH) Cirrhosis of the liver Renal failure	Muscular weakness, cramps, nausea and vomiting, low blood pressure, absence of thirst, Addison's disease, Excessive sweating, Acidosis (metabolic)
<b>Potassium</b> $\text{K}$  (Normal 5 mEq/L)	Muscular weakness and paralysis ultimately cardiac arrest (see page 19) <i>Excessive potassium administration</i> Addison's disease Renal failure	Muscular weakness and paralysis especially respiratory and cardiac (see ECG page 19) Low potassium intake, Starvation, GI obstruction <i>Poor absorption, Steatorrhea, celiac disease, short intestine</i> Excessive potassium loss GI Vomiting, diarrhea, etc. Cutaneous Wounds, burns Renal Tubular defect, diabetic acidosis, metabolic alkalosis Hormonal Adrenal steroids (see page 416) Shift from extracellular to intracellular space Hormonal Testosterone (see page 419) Insulin, glucose Illness, Fasting, periodic paralysis
<b>Calcium</b> $\text{Ca}^{++}$  (Normal 5 mEq/L)	Metastatic calcifications, renal calcinosis Hyperparathyroidism	Tetany Acidosis (metabolic) Hypoparathyroidism Osteomalacia (+)
<b>Magnesium</b> $\text{Mg}^{++}$  (Normal 3 mEq/L)	Depression of CNS and muscle IV administration of magnesium salt	Non-Intoxication (any) possibility

# CORRELATION OF THE SERUM POTASSIUM CONCENTRATION AND THE ELECTROCARDIOGRAM

Proving there is no parallel change in  $\text{K}^+$  and  $\text{Ca}^{++}$



(From Krupp, Sweet, Javel, and Armstrong  
Physician's Handbook, 3rd ed. (Lange Medical Publications))

## Effect of Electrolyte Imbalance on Electrocardiogram

In addition to the serum level of a mineral ion, the ECG may be of value in the diagnosis of ion imbalance, especially if relations occurring during therapy. The use of the compensation relationship that occurs in hypokalemia is reported as of questionable specificity. The most important factors for  $\text{Ca}^{++}$  and  $\text{K}^+$

- A  $\text{Ca}^{++}$**  Calcium deficiency prolongs the Q-T interval. Calcium also shortens the Q-T interval. The quantitation of these changes has not been worked out.
- B  $\text{K}^+$**  The potassium halos are best illustrated by the halo effect. The appearance of the relationship between  $\text{Ca}^{++}$  and  $\text{K}^+$  both clinically in terms of symptoms and ECG effect to their symptoms and ECG effect is antagonistic and a deficiency of one may cancel out the deficiency of the other.
- C  $\text{Mg}^{++}$  and  $\text{Na}^{++}$**  No specific ECG changes have been described for magnesium or sodium ion changes, however the effect is of an opposite and opposite to those of potassium.
- D Respiratory alkalosis** Induced by high concentrations of  $\text{CO}_2$  in inspired air leads to a decreased height of R and T wave, elevation of ST depression of ST-T and a decrease in the amplitude of T, both.

## Relation of Nitrogen to Ion

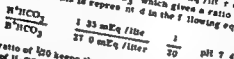
The negative ion is concerned almost entirely with osmotic equilibrium and pH change except for the serum inorganic phosphate. The phosphate appears to be needed for proper utilization (phosphorylation) of glucose and possibly fatty acids and so a definite quantitative relationship in addition to the V-glucose when the latter is indicated.

mEq/l	Normal	Respiratory Acidosis		Metabolic Acidosis		pH
		Compensation	Alk base	Compensation	Alk base	
H <sub>2</sub> CO <sub>3</sub>	1.35	> 1.35	< 1.35	< 1.35	< 1.35	7.35
5						
10						
15						
20						
25						
30						
35						
40						
B <sub>2</sub> CO <sub>3</sub>	37.0	> 37.0	< 37.0	< 37.0	< 37.0	37.0
Sum CO <sub>2</sub>	1.20	< 1.20	> 1.20	> 1.20	> 1.20	1.20
pH	7.35	< 7.35	> 7.35	> 7.35	> 7.35	7.35
Mechanism	Normal	Compensation for loss of CO <sub>2</sub> by blowing from the lungs	Compensation for loss of CO <sub>2</sub> by blowing from the lungs	Compensation for loss of H <sup>+</sup> by loss of H <sub>2</sub> O	Compensation for loss of H <sup>+</sup> by loss of H <sub>2</sub> O	Normal
Ca						
C <sub>2</sub>						
Te	Non					

# ACID BASE REGULATION

31

**Acid Base Equilibrium**  
 The pH of the extracellular fluid during life is maintained at 7.35 to 7.45. A pH beyond this range is incompatible with life. The regulation of the pH within such narrow limits is the function of the buffer systems of the body. Principally the bicarbonate buffer system is composed of 1.35 mEq/liter of  $H_2CO_3$  and 27.0 mEq/liter of  $NaHCO_3$ . This is represented in the following equations:



This ratio of 1:20 keeps the pH at 7.35 regardless of the disturbance of the bicarbonate buffer system. The maintenance of equilibrium through two important physiological mechanisms: the respiratory and the metabolic mechanisms (see page 30).

**A. The Respiratory Mechanism (Lung and Respiratory Center)**  
 Partial pressure of  $CO_2$  in the blood is regulated by increasing or eliminating the volume of  $CO_2$  in the blood.

**B. The Metabolic Mechanism**  
 Since most products of metabolic processes are acids ( $PO_4^{3-}$ ,  $SO_4^{2-}$ ,  $HCO_3^-$  and organic acids), it is important for the body to maintain a balance of  $K^+$ ,  $Ca^{++}$ , and  $Mg^{++}$  to maintain pH at 7.35. The kidneys participate in this conservation through two mechanisms:  
 1. By the ability to secrete an acid urine (maximum about pH 4.5) so conserving relatively more base than acid.  
 2. By the ability of the renal tubule to manufacture  $NH_4^+$  which combines with anion thereby allowing fixed base to be reabsorbed by the renal tubules.

## TREATMENT OF ACID BASE AND ELECTROLYTE IMBALANCE

In the clinical management of derangement of the acid base and electrolyte balance, the attempt is made to return the electrolyte levels to normal. This is accomplished by determining the levels of the important electrolytes ( $Na^+$ ,  $K^+$ ,  $Ca^{++}$ ,  $CO_2$  and  $Cl^-$ ) in the blood and then administering the salts or solutions necessary to return the electrolyte pattern to normal.

With normal renal function it is rarely necessary to be concerned with the individual electrolytes. If fluid of proper osmotic pressure (i.e. isotonic hypotonic or hypertonic as the patient's state demands) is administered, the kidney will adjust the various ions.

Although many formulas have been advocated to calculate replacement of electrolytes, their use is rather limited for most abnormal conditions are mixed types. No formula can yet determine intracellular needs. However, some general principles are assist in determining electrolyte replacement.

## 2. Replacement Calculations

- A. Sodium Replacement (and Cl<sup>-</sup> when used with Na<sup>+</sup> or NaCl)  
 becomes a subject concerned mainly with osmotic pressures and because replacement therapy is used to effect movement of water out of the intracellular compartment the estimation of Na<sup>+</sup> replacement must be made in terms of total body water

$$\text{Replacement of total body water (in litres)} = \frac{\text{mEq Na}^+}{\text{deficiency}} \times \text{Amount of Na}^+ added for replacement$$

Formula: 70 kg muscular individual has a sum Na<sup>+</sup> of 12 mEq/litre

Estimated total body H<sub>2</sub>O = 60% of body weight = 42 litres

Estimated sodium deficiency = 20 mEq/litre

4 x 20 = 80 mEq Na<sup>+</sup> necessary for replacement

Administer half the amount initially the remainder in the next 48 hours. Cl<sup>-</sup> as salt by mouth or hyperosmotic solution

- III. In the case of the replacement of fluid ion, a safe initial replacement can be made especially when parenteral administration is contemplated by estimation of the deficiency in extracellular fluid. This type of calculation is obviously incorrect in the case of K<sup>+</sup> since this ion is largely intracellular but the amount administered can be given quite rapidly without danger of excessive concentration being produced. In the case of HCO<sub>3</sub><sup>-</sup> replacement it also is necessary to take account of the intracellular HCO<sub>3</sub><sup>-</sup>. The same is true of Cl<sup>-</sup> when given to replace HCO<sub>3</sub><sup>-</sup>. For use of calculation this formula assumes an extracellular fluid content equal to 1/3 of the body weight. This estimate is not exact and is especially incorrect in cases of disturbed fluid and electrolyte balance.

$$\text{Amount ion needed (in mEq)} = \frac{\text{Patient's wt (in kg)}}{3} \times \left\{ \text{normal value of ion (in mEq/L)} - \text{Patient's level of ion (in mEq/L)} \right\}$$

Formula: 100 kg man has serum CO<sub>2</sub> content of 15 mEq/L. How much sodium lactate is needed to bring serum CO<sub>2</sub> to normal?

$$\text{mEq of CO}_2 = \frac{100}{3} \times (27 - 15) = 33 \times 12 = 396 \text{ mEq}$$

Therefore 396 mEq of sodium lactate is needed by the patient

Milliequivalent Conversion Factors  
 for Conversion of Blood Chemistry Findings

To find ml mEq/L of	Divide mg % or vol % by
Chloride (from Cl)	3.0
Chloride (from NaCl)	3.5
CO <sub>2</sub> Combining power	5.85
Magnesium	2.22
Phosphorus mill (millimoles)	1.2
Potassium	3.1
Sodium	3.0
	2.5

Table for Determining Quantity of Salt or Salts  
Necessary for Electrolyte Replacement

Salt	Quantity in Grams or Yield	
	25 mEq of cation	100 mEq of cation
NaCl	1.3	5.8
NaHCO <sub>3</sub>	2.1	8.4
Sodium citrate (hyd at 41.1%)	2.8	11.6
Sodium lactate	2.8	11.2
KCl	1.8	7.8
KHCO <sub>3</sub>	2.5	10.0
Potassium citrate	2.8	10.8
MgSO <sub>4</sub> (7.1%)	1.5	6.0
CaCl <sub>2</sub>	1.4	5.5
Calcium gluconate	1.6	6.4
Cs	3.9	15.4
NH <sub>4</sub> Cl	1.4	5.5

After calculating the amount needed, select the solution to be used by referring to the following list of solutions.

#### IV FLUIDS AVAILABLE FOR CORRECTING ELECTROLYTE DISTURBANCES

Many fluids to meet various therapeutic needs have been developed and are commercially obtainable. The following is a partial list of the solutions available based on their electrolyte content. Isotonic and isotonic solutions are those that balance serum available as one tries to be added to other fluids. When these or any other electrolyte solutions are added, be certain the final fluid is isotonic, half isotonic and preferably isotonic or hypotonic when being given I.V. Isotonic solutions are 5% glucose in water, solutions of electrolyte containing about 150 mEq/l of anions and cations per liter.

When the fluid is considered in terms of the milliequivalent per liter of the individual ion, then the appropriate fluid becomes easier to select. The best fluid to correct the disturbances that are present. By thinking of fluids in terms of mEq/liter, one can also deal with solutions as they meet a disturbed electrolyte fluid balance that may occur, e.g., hypertonic NaCl (3.5%) solutions to treat sodium deficiency. Electrolyte data can be located from the formula on page 22. Before selecting the solution, the fluid must also be determined and considered in calculating the total volume needed.



## 24 Intravenous Fluids

### Neutral Solutions Containing Only Sodium and Chloride

- A Isotonic Solution Isotonic Sodium Chloride Solution U S P  
Injection of Sodium Chloride B P (0.85%)  
1.2. Most widely used isotonic solution To replace lost base (Na) and chloride

Constituents	% Sol	Gm per liter	mEq / liter of ions	
			Na	Cl
NaCl	0.85	8.5	145	145

### B Hypotonic Solution (0.45%)

Uses To replace lost base (Na) and chloride and to allow excess water for metabolic needs

Constituents	% Sol	Gm per liter	mEq / liter of ions	
			Na	Cl
NaCl	0.45	4.5	77	77

### C Hypertonic Solutions Variable sodium chloride concentrations (3% and 7% given below)

1.2. To replace sodium and chloride in the treatment of low sodium concentration (see page 22)

Constituents	% Sol	Gm per liter	mEq / liter of ions	
			Na	Cl
NaCl	3	30	515	515
NaCl	7	70	835	835

### Solutions Yielding Free Base Containing Only Sodium

Uses To replace sodium without added anion (usually for cases of metabolic acidosis)

#### A Sodium Lactate

- 1 Concentrate (to be added to other fluids) 1 molar solution 1000 mEq / L (112.3% solution, 112 Gm / L) Concentrate supplied in ampules of 40 cc containing 40 mEq / ampule
- 2 Isotonic Sixth molar sodium lactate

Constituent	% Sol	Gm per liter	mEq / liter of ions	
			Na	Lactate
Sod Lactate	1.87	18.7	167	167

#### B Sodium Bicarbonate

- 1 Concentrate May be injected directly or added to other fluids Supplied in ampules of 50 cc containing 45 mEq / ampule (3.75 Gm in 50 cc solution)
- 2 Sodium bicarbonate may be prepared for injection by adding chemically pure sodium bicarbonate to cool distilled water Do not autoclave or boil solution after  $\text{NaHCO}_3$  has been added 1 Gm  $\text{NaHCO}_3$  12 mEq

Lactate is oxidized to  $\text{CO}_2$  therefore this compound yields free base

Immediate and severe reactions on readministration. Fever, angioneurotic edema, urticarial and other rashes, and periarthritis nodosa may occur.

History of previous administration should be obtained. Cross sensitivity to various sulfonamides may exist. Severe symptoms may be avoided by giving a test dose of 0.5 Gm (7½ gr) and observing for 6 hours.

#### C Precautions

1. Hemoglobin determination and white blood cell count every other day. Discontinue if WBC is less than 6000. Discontinue sulfonamides if granulocyte count is less than 50%.
2. Daily fresh urine for pH (use nitrazine paper) and sediment (increase alkali [sodium bicarbonate] if pH is less than 7.0). Discontinue drug if red blood cells are found in urine (see above). Increase urine output if less than 1500 cc per day or crystalluria occurs (must be examined for in a fresh specimen).
3. Daily observation of patient for drug fever, rash, jaundice, nausea, vomiting, etc.

#### Contraindications to Sulfonamides

1. History of previous hypersensitivity.
2. Renal insufficiency (Very small dose may be used with caution).
3. Liver damage (Proceed with caution if essential).
4. Heart failure (If sulfonamides are absolutely necessary, substitute potassium bicarbonate for sodium bicarbonate as alkalinizing agent).

## PARA AMINO SALICYLIC ACID (PAS)

Para amino salicylic acid (PAS) and its sodium salt have been found to exert considerable tuberculostatic activity. Tubercle bacilli resistant to streptomycin may be susceptible to PAS and vice versa. The simultaneous administration of PAS and streptomycin delays the emergence of strains of tubercle bacilli resistant to the latter. In addition to the bacteriostatic effect, a diuretic activity is present.

PAS is absorbed readily from the gastrointestinal tract. Peak serum concentrations are reached in 30 to 60 minutes and minimum levels are again reached in 4 hours. PAS may be administered orally, intravenously, and subcutaneously.

#### Dosage

- A. Oral. 2 to 3 Gm (30-45 gr) every 6 hours.
- B. Intravenous. 15 Gm in 5% solution given in 2 doses 6 hours apart. 5 mg of heparin should be added to each lot.

#### Toxicity

No side results due to the drug (fever, dermatitis, crystaluria, hematuria, and hypoprothrombinemia) may be observed. Gastrointestinal symptoms may apparently be avoided by parenteral administration of sodium PAS.

Constituent	% Sol	Gm per liter	mEq / liter of ion			
			Na <sup>+</sup>	K <sup>+</sup>	Cl <sup>-</sup>	Cl <sup>-</sup>
NaCl	0.84	8.4	145			145
KCl	0.03	0.3		4		4
CaCl <sub>2</sub>	0.033	0.33			4	4
Total			145	4	4	153

- B Concentration of Total Ions, Magnesium, Calcium (KMC<sup>®</sup>) (To be added to half isotonic or more or enteral fluids) (Con-  
tain 25 mEq K 10 mEq Ca<sup>++</sup> 10 mEq Mg 45 mEq  
Cl / 10 cc ampule or

KCl	1.8 Gm
MgCl <sub>2</sub>	45 Gm
CaCl <sub>2</sub>	55 Gm

In 10 cc

### 5. Types of Mixtures: Electrolyte and Fluids

1. Co-isotonic and parenteral replacement III required simultaneously

- A Low Potassium, Low Free Base Lactated Ringer's Solution  
U.S.P. - Contains in solution of sodium Lactate Base  
(Hartmann's) (isotonic)

Constituent	% Sol	Gm per liter	mEq / liter of ion			
			Na	K	Cl	Cl
NaCl	0.8	8.0	16			16
KCl	0.03	0.3		4		4
CaCl <sub>2</sub>	0.02	0			3.5	3.5
Sol. I total	0.9	8.0	16			
Total			16	4	3.5	10.5
Free Na			16			

- B High Potassium High Free Base Darrow's solution (KAL)  
(isotonic)

Constituent	% Sol	Gm per liter	mEq / liter of ion			
			Na	K	Cl	Lactate
NaCl	0.4	4.0	70		70	
NaCl + KCl	0.6	6.0	53			53
KCl	0.27	2.7		35	35	
Total			123	35	105	53
Free Na			53			

- C Various Intravenous Solutions containing less K<sup>+</sup> and less free base are also available - Gastric Electrolyte Solution  
With 10% Dextrose (Baxter) (isotonic)

Constituent	% Sol	Gm per liter	mEq / liter of ion			
			Na	K <sup>+</sup>	Cl <sup>-</sup>	Lactate
NaCl	0.51	5.1	88		88	
KCl	0.09	0		12	12	
Sol. I total	0.56	5.6	88			50
Dextrose	(10)	(100)				
Total			128	12	100	50
Free Na			50			

Sodium lactate is equivalent to free base

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1. stop

## 23 Venoclysis

per hour by use of 1 needle or 500-1000 cc (1/2-1 qt) per hour with 2 need. A usual practical maximum is 3000-4000 cc (3-4 qt) per day. Care must be taken to avoid overdistending the tissue. This can cause avascularity which may lead to tissue necrosis. When giving glucose solutions 2 1/2% in half normal saline is preferred. 5% may be given in water but not to debilitated elderly patients. Never give in concentrations over 5% glucose to any patient.

To facilitate absorption hyaluronidase may be used. 150 viscosity units are used per 500-1000 cc of fluid. The material may be injected into the subcutis of the hypodermoclysis site into the site of insertion of the needle or may be dissolved in the solution. The rate of fluid absorption is increased up to about 11 times.

## VENOCLYSIS

The intravenous is the route of choice for extra alimentary administration of fluids and nutrients. Fluids, electrolytes, glucose and protein can be administered by this method. The following are the normal requirements.

### Water

The only way water can be given I.V. without electrolytes to supply the obligatory needs of the body is as a solution of 5% or 10% glucose or fructose in distilled water. NEVER administer plain distilled water I.V. Fluids in hydrolyzates may contain significant quantities of electrolytes (NaCl usually) and the end products give rise to urea (see page 12).

### Electrolytes (Minerals)

A. Sodium Sodium chloride (NaCl) is the most important electrolyte. Average daily intake is 3 to 5 Gm (45-75 gr). Average daily requirements 500-1000 cc (1 pt-1 qt) of physiological (0.9% NaCl) saline. If conditions leading to excessive NaCl loss are present added salt may be given. Otherwise do not give over 1 liter (1 qt) physiological saline (8.5-9.0 Gm) daily.

B. Potassium If parenteral therapy is to be continued for over 3 to 5 days one should supply more complete electrolyte replacement especially potassium losses. Average daily basal potassium requirement is about 3 to 4 Gm of KCl (25-50 mEq of potassium). This may be done by use of solutions containing potassium or by adding potassium chloride to saline or glucose (see page 13). Potassium solutions must be administered slowly (~5 mEq/liter of K<sup>+</sup> in ~3 hours). Never administer potassium I.V. in the presence of poor renal function.

### Glucose

May give as 5% or 10% solution. Preferably administered in distilled water but may be given in saline. Never give more rapidly than 0.8 Gm per Kg (1 dr/10 lb) per hour. This is maximum rate of utilization. When given more rapidly than this glycosuria and concomitant fluid loss usually result. In cases where caloric need is great and fluid restriction is necessary more concentrated glucose (20-50%) may be given I.V. very slowly.

Insert 3 f

2.5 parts glucose and fructose have recently been shown to be utilized somewhat more rapidly than glucose and so can be administered more rapidly. The difference however is not marked.

Protein

A. Am. 2 A 14. Usually given as Protein Hydrolysate 5 N N R and usually administered as 5% solution (usually in 5% glucose solution). Higher concentration of protein may be used but the solution is then often too thick or irritating. Solution must be administered slowly about 1 liter (1 qt) in 2 hours. The materials are prepared with varying electrolyte concentrations (one minimal) by different companies and the concentration should be checked if electrolyte restriction is important.

1. Definite contraindications to intravenous protein hydrolysate (Elman)

- Solution which has been open for over 1 hour or is not refrigerated should not be used.
- Untoward reaction of allergic type. Urticaria, angio-neurotic edema, skin rashes. (Transfusion reactions or vomiting are not contraindications. Nausea and vomiting are usually due to too rapid administration of the protein hydrolysate.)

2. Questionable contraindications. Postoperative anuria

B. Citrat 4 Norm 11 m Pl m. (19 P) has about 1% protein. It is an excellent source of protein which can be administered freely. Plasma contains sodium chloride in the same concentration as physiological saline so limit usually is 1 liter (1 qt) per day. The principal advantages of plasma are the danger of producing homologous serum jaundice (even if treated by ultraviolet irradiation) and the high cost of the material.

C. Norm 11 m Serum Albumin U S P or Salt poor Serum Albumin. An excellent source of protein. 25 Gm albumin per 100 cc solution is equivalent in osmotic pressure to 500 cc of plasma. This is an excellent way to administer protein in a small fluid volume and with low salt intake by giving the salt poor material. Albumin is very expensive.

General Indications of Intravenous Alimentation

A. Continuous parenteral feeding should be started. It is usually impossible to administer adequate calories by intravenous means. The following chart outlines the general practical daily physiological limits of intravenous alimentation. The principal limiting factor is the fluid intake. The administration of 3000 cc of 5% protein hydrolysate in 5% glucose solution would give the following amounts of fluid, electrolyte and nutrient material.

Fluid	Mineral (as NaCl)	Glucose	Protein Hydrolysate	Calories
3000 cc	Up to 80 Gm.	150 Gm	150 Gm	1200

Each Gm protein hydrolysate may contain up to 2 Gm NaCl although most products now contain less NaCl.

## Chapter 3

# GENERAL SYMPTOMATIC TREATMENT

## TREATMENT OF CONSTITUTIONAL SYMPTOMS

### PYREXIA (Fever) (code No. 003)

Measures specifically directed toward depression of an elevated body temperature are usually not indicated except for high and prolonged fevers.

#### A. Removal of the Specific Cause of the Fever

1. Infections. See individual specific diseases.
2. Drugs or chemicals. Many drugs (e.g., sulfonamids) are capable of inducing febrile reactions.
3. Dehydration. Provide adequate oral or parenteral fluid.
4. Impairment of C.N.S. heat regulating center. This poses a difficult therapeutic problem. Provide for optimal oxygenation and hydration of tissues and prevent excessive hyperthermia by artificial measures if indicated (see below).

#### B. Reduction of the Fever by Means of Agents. When the body temperature is greater than 40°C (104°F), particularly if prolonged, the following measures may be utilized:

1. Increased fluid intake. By oral or parenteral routes.
2. Warm alcohol sponges. Cooling is due to evaporation.
3. Warm or tepid baths. These cause peripheral vasodilatation.
4. Cold sponges. Provide prompt cooling of skin and psychological relief but interfere with heat loss.
5. Ice bag. Provide local comfort, e.g., for headache.
6. Antipyretic drugs. Quite effective in reducing fever and have a simultaneous antipyretic and analgesic effect. They have the disadvantage that they obscure the clinical picture and may cause undesirable side effects such as diaphoresis, skin eruptions, hematologic changes, nausea and vomiting, cardiovascular depression, etc. Such drugs therefore are to be employed cautiously in infectious fevers and are preferably not used in the enteric fevers (e.g., typhoid fever). The following antipyretic drugs administered every 4 hours per os are among the most commonly used and are probably least apt to produce untoward reactions:

- a. Acetylsalicylic acid (aspirin) 0.3-0.6 Gm. (5-10 gr.)
- b. Sodium salicylate 0.3-0.6 Gm. (5-10 gr.)
- c. Acetophenetidin (phenacetin) 0.3-0.6 Gm. (5-10 gr.)





## II Shock

- 1 **Body position** Place patient in the shock position (see page 3) unless he has a head injury
- 2 **Maintain an adequate airway**
- 3 **Body warmth** Keep the patient comfortably warm. Avoid chilling or excessive externally applied heat since this will further dilate the peripheral vessels.
- 4 **Pain** Control pain (particularly if severe) promptly by the use of appropriate first aid measures and analgesic drugs. Give morphine sulfate 10-30 mg ( $\frac{1}{2}$ - $\frac{1}{2}$  gr) subcut for pain but remember that subcut absorption is poor in patients in shock. In case of severe pain morphine sulfate 10-15 mg ( $\frac{1}{2}$ - $\frac{1}{2}$  gr) i.v. may be used in great advantage. Do not give morphine to unconscious patients or to those who have head injuries or those with respiratory depression. Avoid overdosage with morphine. Substitute barbiturates and salicylates for sedation and analgesia whenever possible.
- 5 **Allay apprehension** by reassuring word and action. Pentobarbital sodium 0.1 Gm ( $\frac{1}{10}$  gr) orally or 0.13 Gm (2 gr) subcut or by rectal suppository may be of value.
- 6 **Intravascular fluid therapy** Replace and maintain adequate blood volume. Need for this may be obtained by the history, vital signs and hematocrit studies. The clinical determination of effective blood volume is difficult however and is subject to considerable variation. There is no single technique or rule upon which to judge the fluid requirements. Response to therapy is a reliable index.
  - a **Saline or glucose solutions** Give immediately 500 cc physiological saline or 5-10% dextrose solution or 200 cc of 5% physiological saline solution (may be given rapidly i.v. while making preparations for plasma, serum, albumin or whole blood). Plasma, serum, albumin and whole blood exert a more sustained increase in blood volume through the colloidal osmotic pressure effect than do dextrose or electrolyte solutions.
  - b **Plasma or serum albumin** Any of the various plasma preparations such as dried or frozen plasma or serum albumin may be employed depending upon their availability. Plasma is most readily procurable, may be rapidly set up for administration and does not require preliminary blood typing. The quantity of plasma to be given depends upon the stage of shock and the response to therapy based upon both clinical and laboratory studies.
    - (1) **Impending shock** Administer 500 cc plasma immediately and follow closely clinically and with hematocrit studies to determine need for further plasma.
    - (2) **Early or advanced shock** Administer 500 cc plasma immediately and repeat with 500 cc every half hour up to a total of two liters depending upon clinical course and hematocrit findings. If shock persists following such therapy the prognosis is very poor.
  - c **Whole blood** If plasma is unavailable or if anemia is present whole blood may be administered as needed.
  - d **Plasma expanders** Evidence during the last few years supports the view that there are several effective plasma

substitute for emergency treatment of shock. These agents have high molecular weights, high oncotic pressures, and the necessary viscosity. They have the added advantage of not causing infectious hepatitis.

- (1) Protins e.g. gelatin 5% solution
- (2) Carbohydrates e.g. dextran 6% solution
- (3) Synthetic polysaccharide

(PVP macro) e.g. polyvinylpyrrolidone  
These agents are more effective in hypovolemic shock without associated decrease in blood volume (e.g. spinal anaesthesia syncope acute intoxication) although they may be employed in severe shock due to any cause.

a. Levartanol (Larotrol) N.N.R. (Levophed) Considered to be very effective but great care must be taken to avoid extravasation. The drug is best given by continuous infusion of a solution containing 4 mg of Larotrol in 100 ml of 5% dextrose. The initial solution should contain 4 mg per litre. The infusion rate should be adjusted to maintain a rapid flow is necessary to produce a desired effect.

If a rapid flow is necessary to produce a desired effect, so that 20-40 drops per minute may be given to stabilize the blood pressure at the required level without administration of too large a total fluid volume. After several hours the flow may be reduced gradually to determine whether it is necessary to continue the drug. The infusion has been used effectively in some patients for a number of days with a significant reduction in mortality from shock.

b. Phenylephrine hydrochloride (Neo Symphephrine Hydrochloride) 0.5 mg i.v. or 5 mg i.m. repeated in an hour if necessary to maintain the blood pressure above 90 mm Hg. If necessary to maintain the blood pressure above 90 mm Hg, the rate of administration by the response of the patient.

Mephentermine (Wyamine) 5-20 mg i.v. given in small increments by the response of the patient. 5-20 mg i.v. given in small increments by the response of the patient.

I.M. repeated in 30-60 minutes as needed to maintain the systolic pressure at 100 mm Hg or in previous hypotensive patient at 120 mm Hg.

B. Specific (Definitive) Measures

1. Anoxia (hypoxia) Anoxia is probably present as a primary complication in all types of shock. Therefore administration of oxygen may be considered for most patients in shock. In some patients in shock oxygen may be administered for other reasons (cardiac pulmonary, etc.) however the patient in impending shock is apprehensive and the oxygen tent soon may become unbearable.

2. Acidosis Not frequently recognized when the clinical and laboratory data indicate coexistence of hypoxia with metabolic acidosis. 500-1000 cc of 5% sodium bicarbonate solution i.v. or by hypodermoclysis as soon as patient can swallow gives fluids.

mouth. Unless there is specific clinical or biochemical evidence of sodium deficiency, avoid administration of more than one liter of physiological saline on the first day. Subsequent parenteral fluids may be given as dextrose solutions. Follow the principles of fluid administration mentioned on pages 10-13.

4. **Adrenal cortical failure.** Adrenocortical steroid therapy has been found to be effective in shock-like states associated with serious medical emergencies. Although treatment is most specifically applied to shock of Addison's crisis, it may also be of spectacular value in certain acute allergic emergencies and severe poisoning intoxications. Hydrocortisone (free alcohol or infusion concentrate for I.V. use) 100-150 mg. daily in 1000 cc. 5% glucose or saline by slow I.V. drip.

5. **Cardiac failure.** Digitalis and other treatment for cardiac failure are indicated only for those patients with pre-existing or presymptomatic occurrence of cardiac failure (see page 182). Digitalis is of no value in shock due to any other cause.

6. **Infection.** Immediate measures should be taken to combat infection if present. Overwhelming infections are capable of producing sufficient metabolic changes in the body tissues to predispose to shock. Prophylactic use of antibiotic drugs in pre-shock or shock patients who are potentially threatened with severe infection (e.g., burn patients) is recommended.

7. **Hemorrhage and anemia.** Although plasma is usually given as an emergency measure in shock complicating hemorrhage, acute anemia must be corrected by replacement with whole blood to prevent hypoxia. The quantity of whole blood to be given will depend upon hematocrit studies.

C. Evaluation of Emergency Therapy. Constant observation of patient is imperative. The pulse, respiration, temperature (rectal) and blood pressure should be evaluated immediately and every 15-30 minutes or oftener thereafter until there is definite improvement of the peripheral circulation.

1. **Rapid recovery.** If vital signs return rapidly to normal keep patient under close observation but withhold further anti-shock therapy. Check vital signs every half hour. Perform hematocrit studies if there is any suspicion whatever that secondary shock exists. Remember that hemoconcentration usually precedes blood pressure and pulse changes. After eliminating potential or existing shock-producing factors, the patient may be managed expectantly until it is reasonably certain that danger has passed.

2. **Prolonged recovery.** If the vital signs remain abnormal for even a brief period after initial measures or show evidence of further progression of peripheral circulatory failure, institute further vigorous anti-shock therapy. Blood hemoglobin, RBC and hematocrit should be determined immediately for a baseline and should be repeated as often as necessary to evaluate the results of therapy.

## INSOMNIA (Sleeplessness) (code No 016)

Insomnia is either a failure to fall asleep frequently or waking from sleep or inability to remain asleep for normal period. Individual sleep requirements however may vary greatly depending upon health, physical activity, training etc. The causes of insomnia are multiple. Emotional or mental preoccupation is the common cause of persistent insomnia.

A. Which are Direct measures towards correction of all factors of sleeping habits (see page 42). It is in relaxation, mental and physical therapy methods could be included under the heading of palliative psychotherapy.

B. General Measures

1. Promotion of optimum environment
  - a. Easily digestible foods in reasonable quantities
  - b. Treatment of existing systemic disease
  - c. Adequate rest and recreation activities and exercise
2. Relief of annoying symptoms which interfere with sleep
  - a. Pain (all types) and Pyrexia      g. Nasal obstruction
  - b. Pruritus      e. Diarrhea      h. Dyspnea and orthopnea
  - c. Hiccups      f. Cough      i. Laryngeal disturbance
3. Quiet pre-bedtime activity. Retention of exciting activities especially in the pre-bedtime period is an individual matter. It is probably advisable for susceptible individuals to avoid listening or thought provoking reading, games, drama or movies for a period of 1 to 2 hours before bedtime. Encourage light reading and other non-stimulating activities.
4. Retention of timetaking habits and drugs especially after 5:00 p.m. e.g. tobacco, coffee, alcohol, phedrine like drug and amphetamine compounds.
5. Provision of adequate sleeping facilities e.g. a comfortable bed and a quiet and dark room with a suitable ventilation temperature and humidity.
6. Warm bath before bedtime may have a relaxing effect.
7. Warm milk taken at bedtime may also have a relaxing effect.

C. Hypnotic Agents The routine use of hypnotic drugs to control insomnia is not only improper but may also be dangerous because of habituation, disturbance of liver and kidney function, etc. The following agents may be used in accordance with individual needs.

1. Wine (sweet sherry or similar) 60 ml (2 to 3 oz) orally at bedtime.
2. White glycerol 30 cc (1 oz) diluted with water orally at bedtime.
3. Phenobarbital U.S.P. Phenobarbital B.P. orally has a low (30 minute) action and exerts a prolonged effect (8 hours) and palliates the patient's hangover. It is contraindicated by kidney and liver disease, contraindicated in children and insufficiently.
- a. Phenobarbital U.S.P. Phenobarbital B.P. 0.1 to 0.2 Gm (1 1/2 to 3 grains) orally 3 times a day.
- b. Phenobarbital Elixir U.S.P. 15 to 30 cc (4 to 8 dr) in 15 mg per 4 cc orally 3 times a day.
- c. Phenobarbital Sodium U.S.P. Phenobarbital B.P. 0.085 to 0.1 Gm (1 1/2 to 2 grains) as 10% solution.

- 4 **Pentobarbital Sodium U S P** Pentobarbitone Sodium, B P orally has a more rapid effect (15-30 minutes) and shorter duration of action (4-6 hours) than phenobarbital. It is excreted by the liver and is therefore contraindicated in hepatic insufficiency.
- a **Pentobarbital Sodium U S P** Pentobarbitone Sodium B P 0.1-0.2 Gm ( $1\frac{1}{2}$ -3 gr) orally h s p r n
- b **Pentobarbital Sodium Elixir (N C A)** 16-32 cc (4-8 dr) contains 30 mg per 4 cc orally h s p r n
- c **Pentobarbital Sodium rectal suppository (N C A)** 0.13 Gm 1-2 inserted rectally h s p r n
- d **Pentobarbital Sodium Sterile U S P** 0.5 Gm ml administered as a freshly prepared 5% solution, I M or I V (slowly and not more than 1 cc per minute)
- Toxic reactions to barbiturates include excitement and delirium (especially in children and in elderly debilitated or febrile patients), drug addiction, barbiturate dermatitis, circulatory depression, and respiratory depression.
- 5 **Chloralhydrate U S P** B P (12.5% Sol) 2-4 cc (0.25-0.50 Gm) orally h s p r n
- 6 **Sodium Bromide Elixir N F** (17.3% Sol) 1-2 dr h s p r n
- 7 **Paraldehyde U S P** B P A useful agent since the clean stock solution is sterile and can be used for oral, rectal, I M or I V administration as needed. The drug has an unpleasant odor. It may be used in 4 lithium.
- a Oral 4-16 cc (1-4 dr) in cracked ice with milk, fruit juice, or whiskey
- b Rectal 16-32 cc (4-8 dr) in 30-60 cc (1-2 oz) of a vegetable oil (1 to 2:1 dilution)
- c I M 3-10 cc (1-2½ dr) (Preferably deep in buttocks)
- d I V 1-3 cc (15-30%) very slowly **CAUTION** May cause respiratory arrest or pulmonary edema

### PAIN (General Aspects) (code No 518)

Concepts of pain and pain mechanisms are highly controversial and for that reason all classifications of pain remain quite arbitrary. For practical purposes there are two principal types of pain: superficial and deep. Pain is sharply localized in superficial structures and diffusely or poorly localized in deeper structures. Deep pain may result in referred pain. Pain may occur as a result of multiple types of stimuli acting upon the various body structures. The relief of pain may be achieved by removal of the stimulus or neutralization of the effects of the stimulus and when these are not feasible by dulling or obliterating the sensation of pain.

#### Analgesic Drugs

Drugs which dull or obliterate pain without producing loss of consciousness (cf. narcotics)

A Salicylate The most commonly used of the various analgesics and is frequently employed for self-medication.

1 Actions and Indications They are antipyretic, analgesic, antirheumatic, and uricosuric in action and are useful in

relieving myalgias neuralgias arthralgias headache and dysmenorrhea

## 2 Preparations dosage and administration are as follows

a Acetylsalicylic Acid U S P B P (aspirin or ASA)  
plain or enteric-coated 0.3 Gm (5 gr) tablets Ordinary dosage is 0.3 to 0.6 Gm (5 to 10 gr) every 4 hours per os 0.3 Gm (5 gr) every 2 to 3 hours is stated to be more effective and results in fewer untoward reactions than larger doses at less frequent intervals. The plain preparation may cause gastric distress which may be avoided by administration of the drug on a full stomach or with 1/2 teaspoon of baking soda or other antacid. The enteric preparation is slow acting but it prevents gastric irritation and is also useful for those patients who might be skeptical of the analgesic value of ordinary aspirin. In certain cases it may be necessary to administer the powdered aspirin rectally in a thin starch paste.

b Sodium Salicylate U S P B P plain or enteric-coated, 0.3 to 0.6 Gm (5 to 10 gr) every 4 hours per os

c Acetylsalicylic acid compound (aspirin compound or APC), a synergistic combination

Acetylsalicylic acid 0.33 gr tils

Phenacetin 0.16 gr tils

Caffein 0.033 gr

Sig 1 tablet is every 3 to 4 hours per os

d Aspirin and codeine preparations (see below)

e Analgesic sedative mixture

Sodium salicylate 10 to 30 til iv

Ethyl or phenobarbital q.s. ad 120 til

Sig 1 to 2 p very 4 hours per os

f Pentobarbital sodium 0.033 gr ss

Acetylsalicylic acid 0.33 gr v

Sig 1 capsule every 4 hours per os

f Methyl Salicylate (Oil of Wintergreen) U S P For external use as a liniment to sore muscles or joints. A 10% preparation in oil of olivum.

3 Untoward reaction Usually mild consisting of sour stomach and dizziness but large doses may cause tinnitus, drowsiness, blurring of vision, nausea and vomiting, diarrhea, disphoria, headache and delirium. In extreme cases liver failure may cause icterus and acute laryngeal edema.

B Acetophenetidin U S P Phenacetin B P 0.3 Gm (5 gr) every 4 hours may be employed in certain cases of salicylate intolerance. In general, however, this drug is more toxic than other analgesic preparations and prolonged use is not advised. Its principal value is in analgesic combination (e.g. APC).

C Codeine U S P (see Gout page 321)

## Narcotic Drug

Drug which relieves pain and the action produces euphoria, sleep or stupor. Pain relief occurs prior to loss of consciousness. Indicated for relief of pain of degree greater than that by analgesic or when pain is of type not susceptible to analgesic drugs (e.g. visceral pain). All of the following drugs

## 25 Narcotics

name to prevent loss. These drugs should always be discontinued as soon as they need for 1 or 2 days.

### A Codeine Phosphate U.S.P. B.I.

1 Actions and indications Pharmacologically similar to morphine but of lesser intensity. C.N.S. depression in ordinary dosages but C.N.S. stimulation in higher dosages diminishes cough reflex decreases bowel motility (constipating). Preferred over morphine for relief of moderate degrees of pain because it is much less habit forming, is much safer and results in fewer untoward reactions.

2 Preparations Dosages and modes of administration

a Codeine Phosphate U.S.P. H.P. 0.008 to 0.065 Gm

(1/4 to 1 gr) orally or subcut every 3-4 hours p.r.n.

Ordinarily 0.065 Gm (1 gr) is ineffective for analgesia; use strong narcotics since larger doses of codeine are attended by untoward side reaction.

b Codeine and acetylsalicylic acid (codein and aspirin)

Codeine phosphate 0.008 to 0.065 gr 1/8 to 1

Acetylsalicylic acid 0.3 to 6 gr v.s.

Sig 1 tablet every 3-4 hours p.r.n.

Codeine and acetylsalicylic acid compound

Codeine phosphate 0.016 to 0.065 gr 1/4 to 1

Acetylsalicylic acid 0.250 gr 1/4 to 1/2

Phenacilin 0.160 gr 1/4 to 1/2

Cafoine 0.032 gr ss

Sig 1 tablet every 3-4 hours p.r.n.

3 Untoward reactions Allergic reactions such as urticaria, pruritus, contact dermatitis and even anaphylactic responses may occur. Addiction is much less apt to follow use of this drug than use of morphine.

### II Meperidine Hydrochloride, U.S.P. Pethidine Hydrochloride

H.I. (Demerol<sup>®</sup>, Dilantin<sup>®</sup>) 0.050 to 0.100 Gm (3/4 to 1 1/2 gr)

orally or I.M. (not subcut) every 3-4 hours p.r.n. is stated to be especially useful for pain associated with smooth muscle spasm (except biliary spasm) although this is disputed. It may be given to individuals who do not tolerate morphine and is less apt than morphine to cause nausea, vomiting, and respiratory depression. Analgesic effect is less than with morphine.

Dilaudid<sup>®</sup> and methadone. Addiction tendency definitely exists.

### C Methadone Hydrochloride, N.N.R. (Amidon<sup>®</sup>, Dolophin<sup>®</sup>)

0.005 to 0.010 Gm (1/2 to 1/8 gr) subcut or I.M. every 3-4 hours

p.r.n. provides analgesia of a level similar to morphine but is slower acting and has a more cumulative action. It is stated to be especially useful for the relief of chronic pain because analgesic tolerance develops more slowly than with morphine. Untoward reactions to the drug are similar to those due to morphine and the addiction tendency is about the same. The drug is not tolerated well orally.

### D Morphine Sulfate This drug remains the most valuable of the potent narcotics for general clinical use.

1 Actions and indications Central nervous system depression resulting in powerful analgesia associated with sedation, euphoria and hypnosis. Selective respiratory center depression and dulling or abolition of the cough reflex. It increases intracranial pressure. Has marked emetic and emetic





## 42 Psychotherapy

psychotic complaints. Remember that somatic complaints of bizarre character are frequently encountered in both neurosis and psychosis.

- 1 General behavior. Appearance of speech, action and attitudes.
- 2 Emotion (mood). Anxiety, agitation, elation or depression.
- 3 Thought content. Illusions and delusions or hallucinations.
- 4 Insight. Judgment, orientation, memory and intelligence.

### General Treatment

A Treatment of Psychosomatic Symptom. Relief is often obtained by treating the symptom. It may be advisable to give relief by treating symptoms. If possible, use of psychotherapy. Rapid response to anti-anxiety and anti-psychotic drugs may be utilized to point out susceptibility to treatment and functional nature of disease. A careful evaluation of rationality of the therapy should follow using simple and understandable terms. A most useful remedy for this purpose is:

B Tincture of Hyaladonna 100 u.s.

Elixir of Phenobarbital q.s. ad 100 i.

Sg. 1 t p t i d 0 min a

B) Planning of Hygienic Living Regimen. Proceed for optimal physiological adjustment to facilitate rapid emotional recovery.

- 1 Insure adequate nutrition with regular balanced meals.
- 2 Plan a workable living schedule with allowance for proper exercise, recreation and sleep.

Treatment of Situation 14: Crisis (Acute emotional disorders due to undischarged emotional stress).

A Permit the patient to ventilate his complaints. Give him the opportunity to tell his troubles (emotional catharsis). Leading questions are a considerable time, but it is generally better to allow patient to tell his own story.

B Help the patient correct or alleviate situational factor.

1 Utilize help of social worker or welfare agency if indicated. Contact family or associates of patient when necessary to obtain additional information and effect desired change in environment.

2 Direct advice and assistance toward simplification of personal problems. Change in environment, marital status, occupational status, etc. may at times be impossible and may complicate rather than simplify problems. Help patient find his solution but allow him to make his own decisions.

C Determine the patient's reason for his reaction to his situation. Sometimes if the patient is assisted in facing his problems objectively and rededicated to change in attitude or reaction towards them, some problems may make his life situation more tolerable.

D Utilize sublimating (directing) technique. Encourage patient to develop other interests, sports, hobbies and skills particularly when patient has excess of time for self-preoccupation.

E Use kindly attitude. Reassurance, suggestion, persuasion and even admonition may be useful as the case demands. Avoid reproaching or arguing with patient.

Try to find Deep seated basis (Long standing or chronic motion disorder due to internal conflict and usually dating back to childhood)

A Re-education or re-orientation choices should be regarded to train a psychiatrist. If there are not available simple symptomatic and supportive medical measures offer the greatest consideration.

#### B AVOID

- 1 Avoid brutally confronting patient with a salient aspect of a sexual symptom.
- 2 Avoid premature interpretation of psychiatric details.
- 3 Avoid anger toward patient because of failure to improve.
- 4 Avoid prolongation of psychiatric study and treatment when it is evident that the prognosis is unsatisfactory or dangerous. In a case where it is better to offer the patient to individuals more prepared to pay psychiatric methods.
- 5 Psychanalytic principles are not valid may be indicated by him personally. Demand psychiatric medication may be fought with danger. Neurosis is a barrier of symptoms is a and if these are broken down a motion is a may be precipitated so the patient may have undue stress toward the end.
- 6 Avoid general psychoanalytic interpretation of symptoms (phenomenon of patient's disease).

#### Evaluation of the Dependent Patient as Subject in Risk

The patient is a lawbreaker and a potential subject in the treatment of his attitude and response. Establish doctor in determining his possibility.

#### A Reason to Direct Observation regarding suicidal intent

- 1 The patient is a lawbreaker and a potential subject in the treatment of his attitude and response. Establish doctor in determining his possibility.
- 2 Patient who is in the danger of committing suicide. The patient is a lawbreaker and a potential subject in the treatment of his attitude and response. Establish doctor in determining his possibility.

#### B Doctor-patient Relationship

- 1 If patient is a lawbreaker and a potential subject in the treatment of his attitude and response. Establish doctor in determining his possibility.
- 2 If patient is a lawbreaker and a potential subject in the treatment of his attitude and response. Establish doctor in determining his possibility.

#### C Patient's Personality and Emotional State

- 1 A patient who is a lawbreaker and a potential subject in the treatment of his attitude and response. Establish doctor in determining his possibility.
- 2 The patient who is a lawbreaker and a potential subject in the treatment of his attitude and response. Establish doctor in determining his possibility.

D An indication of the symptom may be indicated by the patient is not likely to commit suicide. The patient is a lawbreaker and a potential subject in the treatment of his attitude and response.

#### 40. Type of Diet

When preparing the food always make the serving attractive, light in color and smell and serve at the proper temperature. The best prepared diet is one which is often by the patient.

#### 5. CALCULATE THE BASIC CALORIC NEEDS IN STEP 1

After having calculated the basic caloric needs in Step 1, use the following table to be placed at the type of diet for the disease in question. The relative deficits of the diets will be found on pages 52 to 53.

### THE PRINCIPAL TYPES OF DIETS

Disease or Disorder	Diet
Gastrointestinal Peptic ulcer Functional disorders	Moderated Starch High fluid residue soft consistency non-stimulating
Cardiac Liver disease Circulation	Low fat and non greasy High protein high CHO High fluid
Cardiac Congestive failure Hypertension	Low sodium (see ) Low potassium (less than 300 mg /day)
Diabetes	Usually high protein with moderate CHO restriction (see page 55)
Obesity Weight loss and malnutrition	Low calories adequate protein High caloric high protein high vitamin
Renal Nephritis	Low but adequate protein 0.5 Gm /Kg (0.25 Gm /lb ) body weight per day plus total salt/min lost in urine
Allergic Food allergy	Special limitation

These diets vary in the number of calories and/or in the amount of one or more of the dietary components. The next step is to calculate the variations.

#### 5. CALCULATE THE VARIATION OF THE DIETARY COMPOUNDS AS SPECIFIED BY THE DIET

After determining the basic caloric needs and selecting the type of diet, use the following table to calculate the number of calories and the amount of each dietary component for the diet. The remainder of the total calories not supplied by the fixed components of the diet may be made up with unrestricted foods.

# **VARIATIONS OF DIETARY COMPONENTS**

Component	Average Diet	High or Increased	Low or Reduced
Calories (Energy)	Variation Step 1 page 43	25-35% more calories than for maintenance	25-35% less calories than for maintenance
Protein	1 Gm./Kg. (0.5 Gm./lb.) body wt./day (See Step 3 below)	2-4 Gm./Kg. (1-2 Gm./lb.) body wt./day (300 Gm. is about upper limit)	0.5 Gm./Kg. (0.25 Gm./lb.) body wt./day (See note below)
CHO	50% of calories CHO	75% or more of calories CHO	About 25% of calories as CHO
Fat	About 100 Gm. per day	150-250 Gm. per day	70 Gm. or less per day
Vitamin	Supplied by well balanced diet (see page 45)	As in high vitamin foods or supplements	Not indicated
Minerals			
Sodium	5-20 Gm./day	Above 30 Gm./day	0.2-2.0 Gm./day
Calcium	0.1-1.5 Gm./day	Above 3 Gm./day	0.2-0.5 Gm./day
Note: If calcium is abundant the protein may go as low as 0.3 Gm./Kg. (0.15 Gm./lb.) body wt. per day			

Having formulated the dietary prescription (Steps 1-4) prepare the actual diet by selecting foodstuff from the tables in Steps 5-8 and add on the following page.

The selected food items not only provide the desired dietary components but must also be made to fit the catabolic requirements. Use of the very essential role of protein in the diet as well as the highly variable content of the protein food is indicated to begin the dietary selection with protein food. The CHO and total calories as well as the protein value of the various foodstuffs must be kept in mind.

## **STEP 5 - DETERMINE THE PROTEIN NEEDS AND FOODS TO BE USED**

Proteins are necessary for growth and development and as a source of energy. On Gm. of protein 4 Calories. 100 Gm. of protein during its metabolism may yield about 30 Gm. CHO.

## **RECOMMENDED DAILY PROTEIN ALLOWANCES (N. R. C. 1953)**

	American unit per unit of body weight	
1 Adult male	1.5-2.0 Gm./Kg.	0.7-0.9 Gm./lb.
2 Adult female	1.0 Gm./Kg.	0.5 Gm./lb.
3 Pregnant women	1.5 Gm./Kg.	0.7 Gm./lb.
4 Lactating women	2.0 Gm./Kg.	0.9 Gm./lb.

Most of the protein requirement will be obtained from high protein foods which form the basis of the diet in the diet plan. After determining the amount of protein needed for the diet select the high protein food by the use of the tables on the following page.

## HIGH PROTEIN FOODS

These proteins are interchangeable in the diet. One serving yields about 6 Gm. of protein; however, the total caloric content varies.

Food	Serving	Protein Gm.	Cal. Cal.	Fat Gm.	Total Cal.
1 Egg	1 om. <sup>1</sup> / <sub>2</sub>	6	24	0	75
Milk skimmed	1 c. or glass	6	24	10	85
Milk whole	(200 cc.)	6	24	10	130
Lean meat or fish	1 oz. (1 lb. or 16 oz.)	6	24	0	70
Fatty meat or fish	(30 Gm.)	6	24	0	90
Fresh fowl	1 oz. (30 Gm.)	6	24	0	40
Cottage cheese	1 rounded Tbsp.	6	24	1	30
For salad dressing	1 qt. (1 ltr.)	6	24	0.5	100
French soy beans	1/4 cup	6	24	2	60
Chickpeas	1/2 cup	6	24	1.5	100
Kidney	1 oz.	6	24	2	100

## RELATIVE PROTEIN VALUES OF PROTEIN PORTIONS OF DIETS

Diets of more than 10 Gm. or less than 40 Gm. of protein can be calculated by either adding or dividing these basic portions. The table below is so arranged that proteins for low caloric (low fat) and normal or high caloric diets can be selected.

## PROTEIN PORTIONS OF DIETS

For Low caloric (Low fat) Diet Cal.	For Normal or High caloric Diet Cal.
Yields 40 Gm. or 160 Cal. protein	
1 Egg 75	1 Egg 75
2 Cups skim milk (400 cc.) 130	2 Cups whole milk (400 cc.) 260
3 1/2 oz. meat (lean) 245	3 1/2 oz. meat (med fat) 315
450	650*
Yields 30 Gm. or 120 Cal. protein	
1 Egg 75	1 Egg 75
2 Cups skim milk (400 cc.) 130	2 Cups whole milk (400 cc.) 260
2 Tbsp. cottage cheese 60	2 Tbsp. cottage cheese 60
3 1/2 oz. meat (lean) 245	3 1/2 oz. meat (med fat) 315
310	710
Yields 60 Gm. or 240 Cal. protein	
1 Egg 75	1 Egg 75
2 Cups skim milk (400 cc.) 130	2 Cups whole milk (400 cc.) 260
1 1/2 Cup cottage cheese 240	1 1/2 Cup cottage cheese 240
3 1/2 oz. meat (lean) 245	3 1/2 oz. meat (med fat) 315
690*	840
Yields 70 Gm. or 280 Cal. protein	
1 Egg 75	1 Egg 75
2 Cups skim milk (400 cc.) 130	2 Cups whole milk (400 cc.) 260
1 1/2 Cup cottage cheese 240	1 1/2 Cup cottage cheese 240
5 oz. meat (lean) 350	5 oz. meat (med fat) 450
795	1025

Total calories represent the caloric value derived from the carbohydrate and protein content of the foods listed.

## STEP 6 - SELECT THE CARBOHYDRATE FOODS FOR "THE DIET"

Carbohydrates supply energy and usually constitute the largest part of the diet (about 50%). One Gm. of CHO = 4 Cal. If ad quat CHO are given th proteins are p red a sou c s of e gy. At least 10-15% of the diet must be CHO to prevent ketosis.

A For rough approximation of the CHO content of foods the following figures will suffice. An average serving is approximately  $\frac{1}{2}$  cup cooked or 1 cup raw veg + slices of fruits.

Average Serving	Amount of CHO	Total Calories
Veg + fruit	4-6 Gm	25
Fruit	12-15 Gm	50
Slices bread potatoes		
" bean cereal	15-20 Gm	75

B For closer approximation of the CHO content of food

1 8% vegetable and fruits 100 Gm portion yields 3-7 Gm CHO 1 Gm protein, and approximately 15 Calories

Asparagus (4 stalks)	Cucumber (20 slices)	Spinach (1 c)
Bamboo shoots (3/4 )	Egg plant (2 slices)	String beans (1 )
Bean sprouts (1 )	Endive (1 head)	Summer squash (1 c)
Belt greens (1 c)	Lettuce (1/3 head)	Tomato (1 small)
Boccoli (1 c)	Mashed greens (1 c)	Turnip greens (1 c)
Cabbage (1 1/2 c)	Okra (10 pods)	Cantaloupe (1/4)
Cauliflower (1 c)	Pepper green (1)	Rhubarb (1 c)
Celery (5 stalks)	Radishes (15)	Sweet berries (12)
Chard (1 1/2 c)	Sauerkraut (2/3 c)	Watermelon (1/2 slices)

2 10% vegetables and fruits 100 Gm portion yields 8-12 Gm CHO 1 Gm protein and approximately 40 Calories

Artichoke (1)	Onions (2)	Gooseberries (2/3 c)
Beets (2/3 )	Pumpkins (1/2 c)	Grapefruit (1/2 c )
Carrot (1 1/2 )	Rutabagas (3/4 c)	Honeydew melon (1/10)
Dandelion greens (1 c)	White turnips (3/4 c)	Orange (1 small)
Green beans (1 c)	Winter squash (1 c)	Pineapple (1 large)
Leek (4 stalks)	Cranberries (1 )	Tangerines (2)

3 15% vegetables and fruits (fresh or canned without sugar) 100 Gm portion yields 12-15 Gm CHO 1 Gm protein and approximately 50 Calories

Apple (1 medium)	Currants (1 )	Figs (3/4 c)
Apricots (2)	Grapes (1 c)	Pineapple (2 slices)
Blackberries (1 c)	Loganberries (1 c)	Raspberries (1 c)
Blueberries (2/3 c)	Nectarines (2)	Strawberries (1 c)
Cherries (18)	Pears (1)	

4 High CHO foods Serving yields approximately 75-100 Calories  
a 1/2 c pot of macaroni 1 green corn (1 c) yields 3-5 Gm protein

b 1/2 cup of parsnips and potatoes (1 lb) yields 1 Gm protein

c 1 lb of bread (1 lb) yields 2 Gm protein

d Cereal (1 c) yields 2 Gm protein

(1) 4 oz (3) 5 p t l

(2) 3 graham

(4) 3 Hy kr p<sup>2</sup>

## III Fat and Vitamins

- e Dried fruits 1/4 cup raisins 3 or 4 large prunes or  
dates 1 1/2 large figs (also yields 1 Gm protein)
- f Sugar 3 cubes or 2 heaping spoonfuls

### C Caloric Values of Beverages (For milk see page 43)

Coffee (15 c)	Cal/oz (15 c)
Tea	12
Dry wine	5
Sw. wine	45
Liquor	75

Cal. values derived mainly from a

## THE FAT REQUIREMENTS OF THE BODY AND THE FAT IN FOOD

The fat requirements of the body are unknown, but fat forms an important source of food. 1 Gm. fat = 9 Cal. Fats usually make up the remainder of the caloric intake after the protein and CHO portions have been selected. Most of the protein-containing foods also contain fat, which must be calculated and combined with total fat intake (see page 43).

### Caloric Value of Servings of Pure Fats (Each quantity equals approximately 40 Calories)

1 Tbsp. butter 1 Tbsp. margarine 1 Tbsp. animal fat 1 Tbsp. oil  
1 Tbsp. lard 1 Tbsp. mayonnaise 1 Tbsp. light cream 1 strip bacon  
One square pat of butter or margarine = 100 Calories

## THE FAT REQUIREMENTS OF THE BODY AND THE FAT IN FOOD

These normal daily requirements are adequately supplied by the basic diet shown on page 43. It is only in cases of restricted diets or abnormal metabolic states (e.g., diabetes, fever, thyrotoxicosis, digestive absorption, etc.) that vitamin supplements may be necessary. For therapeutic dosages, see pages 58 to 64.

### DAILY ALLOWANCES OF VITAMINS (N. R. C. 1935)

Vitamin and Daily Requirement	Natural Sources
A 5,000-8,000 I.U.	Vitamin A Milk, butter, and liver oils Carotene precursors Carrots, sweet potatoes, apricots, spinach, thin green leaved vegetables
B <sub>1</sub> Thiamine 1.2-1.6 mg	Yeast, whole grain cereal, and liver, egg yolk
B <sub>2</sub> Riboflavin 1.4-2.5 mg	Milk, yeast, eggs, liver, meat
P P Niacin 10-16 mg	Legumes, meat, rice, bran, whole wheat
C Ascorbic Acid 150 mg	Citrus fruit, green peppers, parsley, tomatoes, cabbage, radishes
D 400 Units	Butter, liver, egg yolk, fish, liver oils

## STEP 9 - DETERMINE THE NEED FOR MINERAL SUPPLEMENTS

Daily requirements of the minerals are supplied in a well balanced diet ( see page 43). Additional amount are required when an abnormal loss or increased demand arises. The greatest mineral is usually then given as ribed as drug. The 2 deficiencies most likely to occur are those of calcium and iron. Iodine deficiency in endemic areas can be prevented if iodine salt is used.

### DAILY ALLOWANCES OF MINERALS (H. R. C. 1953)

Mineral	Allowance	Natural Sources
Calcium	0.8 Gm. for adults 1.5 Gm. for pregnant and lactating women	Milk and milk products (1 Gm. salt milk)
Iron	12-15 mg. for children at weaning women 1-2 mg. for infants	Liver, egg yolk, kidney, beef, whole wheat, green vegetables
Copper	1-2 mg.	Liver, egg yolk, bran, tomatoes
Iodine	0.12-0.3 mg.	Iodized salt, Vegetables, seaweed, iodine rich soil
Sodium	2-5 Gm.	Salt, milk, meat, eggs
Phosphorus	1-1.5 Gm. (2-3 Gm. during pregnancy)	Milk, liver, egg yolk, cereals, nuts, beans
Potassium	1-4 Gm.	All vegetables and fruits

## STEP 10 - ANALYZE THE UNDER FREQUENCY AND TIME OF FEEDING.

In the management of infants it may be necessary to give greater number of larger or smaller feedings per day at regular intervals or the greater portion of the day. The diet is in which the most important is food.

1. Malnutrition, fever, thyroiditis, jaundice. Frequent feeding to increase diet with the
2. Particular. Frequent small feedings to maintain a normal buffering action.
3. Diabetes. Frequent small feedings to maintain a constant blood sugar.
4. Digestive capacity (e.g. postoperative). Frequent small feedings.

## STEP 11 - GIVE DETAILED INSTRUCTIONS TO THE PATIENT

When the diet has been completely planned, carefully planned and fully written instruction must be given to the patient. The should include the proper feeding frequency, number of meals and time of eating. The following description of the diet will aid in formulating the instruction.



## PRINCIPAL TYPES OF DIETS

The following diets are planned around the Basic Foods which form the nucleus of a well balanced diet. See table on page 43.

### Stress Diet

Prevent severe non irritating, buffering diets taken on regular schedule

#### Composition

- Stage I 3 oz. (90 cc.) half milk and half cream (18%) every hour from 7:00 a.m. to 7:00 p.m.
- Stage II Stage I plus 3 feedings of refined cereal (3 oz. per serving) and 1 soft cooked egg daily.
- Stage III Stage II plus creamed soups and pureed vegetables.
- Stage IV 3 oz. (90 cc.) milk and cream every hour plus regular meals of small feedings of lean meat, potato, pureed vegetables, refined cereals and breads, custard, puddings, cream and butter.

Restrictions Meat extracts, bran, raw vegetables and fruits, tea, coffee, condiments, spices, alcohol and carbonated beverages.

### Moenigrahn's Diet

Anticancer diet including peptic ulcers. As now generally employed means frequent feeding of purged foods. Originally described as follows:

- 6 a.m. Tea, white bread and butter.
- 9 a.m. Oatmeal with milk, white bread and butter.
- 1 p.m. Dinner. As much as desired of meat balls, broiled chops, omelet, fish balls, vegetable or meat or fish gratin, mashed potatoes, vegetable purées or soups, creamed vegetables, stewed apricots, applesauce, gruel, and rice and tapioca puddings.
- 3 p.m. Cocoa.
- 6 p.m. White bread and butter, sliced meats, cheese and tea.

### Blind Diet

A normal diet modified to be smooth, non irritating and bland in taste. May also be used as low residue diet.

Composition Lean meats, fish, poultry, egg, milk, potato, pureed vegetables and fruits, refined cereals and breads, custards, puddings, gelatin desserts, cream, butter, margarine, salt and sugar in moderation.

Restrictions Fried foods, raw vegetables, fruits and fruit juices, pickles, condiments, bran, whole grain cereals and bread, carbonated beverages, alcohol and coffee.

### Low Fat, Non-Coffining Diet

Composition Lean meat, fish, poultry, skimmed milk or butter, milk, cottage cheese, cereal products, bread, vegetables and fruits except those listed below, gelatin desserts, sherbet, puddings without cream, sugars and jellies.

Restrictions Pork, ham, bacon, fatty cuts of any meat, cream, cabbage, family onions, turnips, cucumbers, radishes, green peppers, dried beans and peas, melons, raw apples, butter, margarine, mayonnaise, oil, nuts, chocolate, fried foods, pastries and highly seasoned foods.

High Prot in High CHO Low F t Diet

Composition A low f t diet with stress placed on large servings of lean meat, eggs, skimmed milk or buttermilk, cottage cheese, cereal, breads, fruit juices, sugar and jelly. To calculate a definite amount of prot in for this diet see tables on page 42.

Restriction Same as for low f t non gas limiting diet.

High Residue Diet

A normal diet with a maximum of bulk.

Composition All of the basic foods with extra servings of whole grain cereals and bread, raw vegetables and fruits and an adequate amount of fluids.

Restriction None.

Diet Restrict d in Sodium Content

Sodium restricted diets usually employ 1-5 Gm. of sodium (1-75 Gm. sodium chloride) or more. For best therapeutic results diets should contain less than 400 mg. of sodium (1-5 Gm. sodium chloride).

The following two low sodium diet both contain 2,000 Calories. They are the same in composition except for the average

250 mg. sodium diet use Lonslac® as beverage

500 mg. sodium diet use whole milk as beverage

Breakfast

Fruit	1/2 cup
Salt free cooked or puffed cereal	1/2 cup
Salt free bread	1 slice
Salt free butter or margarine	2 tsp. (1 pat)
Egg	1
Lonslac® or whole milk	1/2 cup

Noon and Evening Meal

Salt free ham or	3 1/2 oz.
Salt free potato or	1/2 cup
Salt free cooked or raw vegetables	as desired
Salt free bread	1 slice
Salt free butter or margarine	2 tsp. (1 pat)
Fruit	1/2 cup
Lonslac® or whole milk	8 oz.

Additional Instructions for Restrictions on Fat with L.P. diet

1. Lonslac® is prepared by mixing 1/2 cup dry powdered with 2 cups of water. This may be flavored with chocolate.
2. To make salt free toasting and kneading machine in five change of cold water.
3. Use of frozen vegetables also acceptable. salt free canned vegetables. Myun, rice, corn, beans, carrots, apples and other general winter.
4. Use only fresh cooked fruit.
5. Use lightly salted glazes in salads and dressings.
6. Myun, pepper, herbs and other spices.
7. Myun, one fifth sodium free salt substitute.

## II II selection

- 1 If m bacon bacon fat salt pork corned beef or pork luncheon meat canned meats fish or poultry
- 2 Prepared cereals with salt quick cooking cereals breads seasoned with baking powder or baking soda
- 3 Prepared foods or prepared desserts
- 4 Canned vegetables dried fruits commercial salad dressing catsup
- 5 Salt d nuts salted popcorn potato chips
- 6 Garlic salt onion salt celery salt salt baking powder baking soda
- 7 Celery green pickles relishes chard
- 8 To avoid disease Ca large family onions turnips peppers dried beans cucumbers sweet potatoes raw apples in one

## C Approximate S II m Content of Common Foods (in mg per serving) This list gives the natural content without the addition of salt baking powder or baking soda

- 1 Fresh meat fish and poultry 3½ oz (100 Gm) serving
 

Lamb	78	98	Oyster	73	Chicken leg	110
Pork	98		Cod fish	80	Turkey leg	92
Beef	31		Halibut	88	Chicken breast	78
V al	48		Salmon	48	Turkey breast	40
- 2 Egg 1 40
- 3 Milk 7 oz glass (200 cc) Cultured buttermilk 210 fresh whole milk 110 reconstituted whole milk (Lonalac®) 3
- 4 Cheese 1 oz (30 Gm) Processed 450 cheddar 210 cottage 100 cream 73
- 5 Legumes ½ cup (4 oz or 120 Gm) fresh or ¼ cup (1 oz or 30 Gm) dry Bean and corn 0 1 1 split peas dry 42
- 6 Cereals 1 oz (30 Gm) dry ¼ p whole grain cereals or pasta (macaroni etc) 0 5 4 1 cup dry cold cereals 200 350 puffed cereals 1
- 7 Bread (1 slice) and crackers
 

Commercial bread	180 250	6 Soda crackers	330
Yeast bread without salt	0 3	1 Matzo, plain	0 3
- 8 Vegetables 3½ oz (100 Gm) serving of fresh or frozen (not canned) (For size of serving see page 49)
 

Artichoke	43	Cabbage	5	Endive	18	Potato	
Asparagus	2	Carrots	31	Kale	110	Ski 1 as	0 8
Beans	1 2	Cauliflower	34	Lettuce	12	Pumpkin	0 4
frozen	2	Chard	200	Okra pods	1	Spinach	82
Beta	110	Celery	110	Onion	1	Squash	0 5
Brussels	18	Corn	tra e	Paranips	7	Tomat	3
Brussels	frozen	8	P as	0 8	Turnip	37	
sprouts	18	Eggplant	1	frozen	100		
- 9 Fruit 3½ oz (100 Gm) serving (for size see page 49) Fresh canned and frozen fruits contain less than 10 mg sodium per serving
- 10 Fats 10 Gm (2 tea spoons)
 

Margarine	110	Sweet butter	0 5	Shortening	0 1
Regular butter	98	Oil	0 2	Lard	0 3
- 11 Sweet 10 Gm (2 tea spoons)
 

Sugar	min m amounts	honey	2 0	jelly	0 2
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**II Miscellaneous -**

Beer 8 oz 10      Coca Col ® 1 bottle 10  
 Ginger ale 8 oz 10      N to 1 oz (30 Gm) 0.5  
 Coffee natural herbs and condiments contain only  
 negligible amounts of sodium

**Diabetic Diet**

A calculated diet with regulated amounts of protein and carbohydrate

**1800 Cal Diet**

**Breakfast (7:00 - 9:00 a.m.)**  
 1/2 cup 10% fruit  
 2 egg any style  
 1 tsp butter or margarine  
 1 glass skimmed milk

**Morning Feeding (10:00 a.m.)**  
 1 glass skimmed milk  
 1 inch cube processed  
 or ground Beefsteak

**Noon Meal (12:00 - 1:00 p.m.)**  
 3 oz any lean meat  
 chicken or fish  
 1/2 cup 5% vegetable  
 1/3 cup 5% egg white  
 2 tsp 5% vegetable  
 2 tsp butter or margarine  
 1/2 cup 10% fruit  
 1 glass milk

**Afternoon Feeding (3:00 p.m.)**  
 1 glass skimmed milk  
 1 rounded Tbsp nuts  
 or peanut butter

**Evening Meal (6:00 - 7:00 p.m.)**  
 1 any lean meat  
 chicken or fish  
 1/2 cup 5% vegetable  
 1/3 cup 5% vegetable  
 2 tsp 5% vegetable  
 2 tsp butter or margarine  
 1/2 cup 10% fruit  
 1 glass milk

**Bed Time Feeding (9:00 - 10:00 p.m.)**  
 1 glass skimmed milk  
 1/2 cup (5 amt) cottage  
 cheese

**2500 Cal Diet**

**Breakfast (7:00 - 9:00 a.m.)**  
 1/2 cup 10% fruit  
 2 egg any style  
 2 strips bacon (sp)  
 Coffee or tea as desired

**Morning Feeding (10:00 a.m.)**  
 1 cup whole milk  
 2 inch cube processed  
 or 1/3 cup peanuts

**Noon Meal (12:00 - 1:00 p.m.)**  
 1/2 cup cottage cheese  
 1/2 cup 5% vegetable  
 1/2 cup 10% vegetable  
 1/2 cup 10% fruit  
 2 tsp butter or margarine  
 1 cup whole milk  
 Coffee or tea as desired

**Afternoon Feeding (3:00 p.m.)**  
 1 cup whole milk  
 2 inch cube processed  
 or 1/3 cup peanuts

**Evening Meal (6:00 - 7:00 p.m.)**  
 4 oz lean meat chicken or  
 fish  
 1/2 cup 5% vegetable  
 1/2 cup 10% vegetable  
 1/2 cup 10% fruit  
 2 tsp butter or margarine  
 1 cup skimmed milk  
 Coffee or tea as desired

**Bed Time Feeding (9:00 - 10:00 p.m.)**  
 1 cup whole milk  
 2 inch cube processed  
 or 1/3 cup peanuts

## III Diet

### High Calorie High Prot in High Vitamin Diet

A normal diet containing extra foods high in protein and all of the vitamins

Composition All of the basic foods with increased amounts of meat fish, poultry liver eggs milk cheese whole grain cereals carrots green vegetables citrus fruits butter or margarine (see table on page 48 for high protein foods and table on page 49 for high vitamin foods)

Restrictions None

### Low Calorie Diet

A diet protein, bulky diets which are lower in calories than the patient's daily requirement (see page 43) Amount of food listed in each diet is the total daily intake

#### 1200 Calorie Diet

3 oz lean meat, fish, poultry or cheese  
1 egg  
1 pt skimmed milk or buttermilk  
1 slice bread  
1 serving (1/2 cup) potato or equivalent  
2 servings 10% vegetables  
3-4 servings 5% vegetables  
2 servings 10% fresh fruit  
1 serving 15% fresh fruit  
2 tsp butter or margarine

#### 1600 Calorie Diet

Omit the following from the 1200 Calorie diet  
1 serving potato  
1 serving 10% vegetable  
1 serving 5% vegetable  
1 tsp butter

#### 800 Calorie Diet

3 oz lean meat, fish, or poultry  
2 oz cottage cheese  
1 slice bread  
1 serving 10% vegetable  
3 servings 5% vegetables  
3 servings 10% fruit  
1 pt skimmed milk or buttermilk

#### 1300 Calorie Diet

Add the following to the 1200 Calorie diet  
2 slices bread  
3 tsp butter or margarine  
1 serving 15% fruit

Restrictions All foods candy and beverages except those listed

### Low Protein Diet

A normal diet with the protein foods limited to the minimum but adequate amount

Composition Meat fish poultry legumes eggs milk cereal, bread and nuts limited to give the desired protein intake (see page 48 for protein values) Vegetables fruits fats and sugars may be taken as desired

Restrictions Protein foods in excess of the specified amount

### Special Elimination Diet

A normal diet from which have been eliminated the foods suspected of causing allergic reactions. Such reactions are produced most frequently by wheat eggs and milk less frequently by citrus nuts chocolate and fish. Other foods may infrequently cause reactions

More specialized diets have been prepared by all regimens and are used both diagnostically and therapeutically. Consult books on all regimens for these diets.

### Low Purin Diet

Diet low in nucleoproteins

Foods Forbidden Live kidney sweetbreads sardines anchovies brains whole grain products gravy soups meat extracts asparagus beans cauliflower peas lettuce and mushrooms

Food Restricted All other meat, fish, and fowl

Composition All other foods are allowed. Most protein to be derived from eggs and dairy products

## TUBE FEEDINGS

Tube feedings are employed when swallowing is impossible or painful, the patient is otherwise unable or unwilling to take food by mouth. A convenient means of administering the feedings is with a small polyethylene tube placed intranasally. Many food mixtures may be given; the only requirements are that the food be fluid or in a suspension of very small particles.

Protein hydrolyzates are often irritating. Formulas containing egg tend to occlude the lumina of small tubes. Excellent formula can be prepared by using milk (occasionally lots must be) and lecithin as inate. Lactose<sup>®</sup> trained milk is lactose, sucrose, or glucose. Fat such as cod oil may be added if emulsified with Tween 80<sup>®</sup> or a similar agent. Vitamins and minerals are added as indicated. Examples of tube feeding formulas are as follows:

- 1 Low sodium high protein diet - Supplies 3 000 Calories per 3 000 cc (1 Cal/cc) contains 133 Gm protein  
 Sterilized canned baby meat 400 Gm (1½ cans)  
 Tomato juice 1900 cc  
 Prune juice 90 cc  
 All purpose Soyab<sup>®</sup> 200 Gm  
 Lactose 315 Gm (1½ cup)  
 Water q. d. 3000 cc
- 2 Inexpensive high protein formula 3 000 Calories per 3 000 cc (1 Cal/cc) contains 120 Gm protein  
 Homogenized milk 2200  
 ½ milk and ½ cream 600 cc  
 Eggs 6  
 Dextrose Maltose<sup>®</sup> or lactose 7 Tbsp
- 3 Low sodium high protein formula 3 000 Calories per 3 000 cc (1 Cal/cc) contains 150 Gm protein 78 mg sodium  
 Lactose<sup>®</sup> 600 Gm  
 Water q. d. 3000 cc

## THE VITAMINS

The vitamins are organic substances which are essential for life and which must be supplied to the organism from exogenous sources. They are not amines but appear to function as enzymes or coenzymes in important metabolic processes.

The best and only certain source of all the vitamins is a well-lanced diet. Therefore a healthy person with proper nutrition does not require vitamin supplements, yet many persons, even in good service circumstances, eat less vitamin-containing foods than are necessary for optimal health.

No criterion of deficiency exists to show that vitamins exert a beneficial effect. There is much indiscriminate use of the vitamin supplements.

It has therefore to be considered how variation in the body requires more depending upon age, activity, diet, metabolic rate, and other factors affecting the absorption, utilization, and excretion of vitamins. Vitamin deficiencies are almost always multiple, particularly of fat-soluble or B complex vitamins as a group. Early signs of vitamin deficiency are usually non-specific, vague, and mild and are easily misinterpreted or missed entirely. The crude sources of the vitamins are often more efficacious in therapy than the pure or synthetic. Only during the more severe phases of the deficiency is it usually necessary to resort to the use of pure vitamins. The use of a "pure" vitamin in the face of a true multiple vitamin deficiency may aggravate rather than help the condition. Treatment of vitamin deficiencies requires an adequate, balanced, high protein and high vitamin diet in addition to a necessary vitamin supplement. In general, it is wise to use vitamins therapeutically in 5-10 times the amounts required for daily maintenance.

The Recommended Daily Dietary Allowances listed below are adopted from the recommendations of the Food and Nutrition Board of the National Research Council (Nutrition Reviews 6:319, 1948).

## FAT-SOLUBLE VITAMINS

### VITAMIN A

Vitamin A is necessary for normal function and structure of all epithelial cells and for the synthesis of visual purple for retinal rod functions and hence for vision in dim light. Carotene precursor of vitamin A is converted to vitamin A probably in the liver. Vitamin A is stored in the Kupffer cells in increasing amounts with increasing age up to adulthood. Vitamin A intake is massive (e.g., 500,000 to 1,000,000 I.U. daily) in many cases of alopecia, itching, and bone pain from new growth of periosteal bone. The principal dietary sources are leafy green and yellow vegetables, whole milk, butter, and eggs.

Recommended daily dietary allowances (1 U.S.P. unit = 1 I.U.) are as follows: Adults, 5,000 I.U.; during pregnancy, 6,000 I.U.; during lactation, 8,000 I.U.

## AVITAMINOSIS A (code No 010 761)

Avitaminosis A is due to inadequate intake (especially in children) poor absorption and in some instances failure of the liver to convert carotene to vitamin A

**A Mild or Early Manifestation** Dryness of the skin (night blindness and follicular hyperkeratosis)

**B Severe or Late Manifestations** Xerophthalmia atrophy and keratinization of the skin and keratomalacia

**C Treatment** Deficiency. Dark adaptation is impaired. Low blood level of carotene or vitamin A may be helpful. A therapeutic trial with 25 000 to 50 000 I.U. daily for 4 weeks may also be employed

### Treatment

Of vitamin A U.S.P. Vitamin A B.P. 15 000 to 25 000 I.U. once or twice daily until symptoms improve. If absorption deficit is present, may be necessary to administer the vitamin A or to give the same dosage of vitamin A in oil (1 M (50 000 units/cc in sesame oil) or similar dose of an accepted vitamin A and D preparation. Skin lesions may require more treatment

## VITAMIN D

Vitamin D has two important actions. It is essential in the regulation of calcium and urinary excretion of phosphorus. It is stored in the liver, skin and brain. Dose of vitamin D over 5 000 to 10 000 I.U./Kg of body weight produces symptoms indistinguishable from those of hypocalcemia. The principal dietary sources are butter, egg yolk, fortified milk and fish, but not even these food sources are rich in vitamin D.

Recommended daily dietary allowance (1 U.S.P. unit = 1 I.U. = 0.025 mcg of ergocalciferol) during day (6 to 12 months) pregnancy and lactation 400 I.U. The normal adult allowance is unknown. Most vitamin D is necessary in the absence of ultraviolet light which is obtained naturally or (7-dehydrocholesterol) in the skin. Vitamin D preparation is found only in animal or plant sources is more readily absorbed

## AVITAMINOSIS D (code No 010 764)

Avitaminosis D is usually due to inadequate dietary intake or lack of sunlight or absorption deficit

**A Clinical Finding** Lack of vitamin D leads to tetany, rickets in children and osteomalacia in adults

**B Treatment** Deficiency. Serum calcium and phosphorus may be normal or decreased and alkaline phosphatase is generally increased. Urinary calcium excretion is decreased

### Treatment

**A Rickets** Calcitriol U.S.P. Vitamin D B.P. or

Calciferol U.S.P. (D<sub>2</sub>) or 7-dehydrocholesterol (D<sub>3</sub>)

U.S.P. (D<sub>3</sub>) 1 000 to 2 500 I.U. or similar dose of an accepted



A and D preparation daily by mouth for several months plus adequate milk. Some cases of rickets are exceedingly resistant and require huge doses of vitamin D (50 000-150 000 I.U. per day).

■ Out-comes: 1. (See page 382)

## VITAMIN E

Although vitamin E plays a role in the normal physiology of certain animals, there is no satisfactory evidence of activity in humans. It is relatively non-toxic. It has been used without apparent benefit in some cases of habitual abortion in doses of 50-100 mg α-tocopherol daily. It has also been used without satisfactory results in some neuromuscular syndromes and in heart disease.

## VITAMIN K

Vitamin K is necessary for prothrombin formation by the liver and hence is important for proper blood coagulation. Bacterial synthesis of vitamin K occurs in the intestine. It is not stored in appreciable amounts in the body. Naturally occurring vitamin K is non-toxic but menadione in doses of 180 mg is reported to cause vomiting, porphyrinuria, and transient albuminuria. The principal dietary sources are green leaves of plants, especially spinach, cauliflower, cabbage, and lettuce; egg yolk and soy beans. Daily dietary needs are unknown.

### AVITAMINOSIS K (code No 010 766)

A dietary deficiency of vitamin K probably never occurs. Liver disease may affect the normal synthesis of prothrombin by liver parenchyma or may cause inadequate bile formation which may interfere with absorption. Some drugs (e.g. salicylates) tend to lower prothrombin and thereby to increase the requirements of vitamin K. Avitaminosis is manifested by hemorrhages, especially from mucous membranes or at points of trauma, and by a prolonged prothrombin time.

#### Treatment

- A Liver disease when associated with a lowered prothrombin, should be treated with 2-5 mg Menadione Sodium Disulfite Injection U.S.P. or Mephthone Injection B.P. daily I.V. or I.M. even though the ability of the liver to form prothrombin may be impaired. Menadione 1-3 mg by mouth with 1-4 Gm. bile salts to increase absorption may be used especially in cases of biliary obstruction.
- B Pregnant women generally should receive 2-5 mg menadione sodium disulfite I.V. or I.M. 12-72 hours before delivery to prevent bleeding in newborn infant.
- Dicumarol® toxicity (see page 217)

## WATER-SOLUBLE VITAMINS

### VITAMIN B COMPLEX

The members of the vitamin B complex are very intimately associated in occurrence as well as in function. As a result of this close interrelationship, it is doubtful that a deficiency of a single B vitamin ever exists except under experimental conditions. Deficiency of a single member of the B complex would lead to impaired metabolism of the other. Hence although a certain clinical picture may predominate in the absence of a single member of the complex this does not signify that the deficiency can be entirely corrected by administration of that single factor. Therefore specific therapy must always be applied in the presence of a definite or probable source of all of the other members of the B complex. Water-soluble vitamins should be administered in divided doses throughout the day to prevent excessive loss in the urine.

#### VITAMIN B<sub>1</sub> (Thiamine Hydrochloride)

Thiamine hydrochloride (an uric acid hydrochloride) functions primarily as coenzyme concerned with pyruvic acid metabolism. It is a powerful activator of the citric acid cycle and is biologically active in several other enzyme systems as well. The need for this vitamin varies with the amount of alcohol consumed. It is readily absorbed from the intestine and excreted by the kidney with limited storage in muscle. Liver, kidney, heart and brain.

Recommended daily dietary allowance is 1.2 to 1.6 mg for adult and 1.5 mg during pregnancy and lactation. The principal dietary sources are yeast, the hull of grain, rice bran, peas and peanuts. It is found in the foods that contain ascorbic acid (Stimulating exposure to sunlight at redness the thiamine content of foods).

#### AVITAMINOSIS B<sub>1</sub> (Beriberi) (code No. 010 7621)

Avitaminosis B<sub>1</sub> results from an inadequate intake due to faulty judgements of diet or excessive cooking or processing of foods. The increased need for vitamin B<sub>1</sub> during fever, high CHO intake or thyrotoxicosis may lead to deficiency.

**A Mild or Early Manifestation.** Vague multiple complaints such as tingling, numbness, and incoordination and muscle cramps, tenderness of the legs, peripheral and hyperreflexia, slow heart rate by hypocoactivity of the heart and ankle.

**B Severe or Late Manifestations (B<sub>1</sub> Deficiency).** Severe anorexia, polyneuritis, oedema, subcutaneous oedema, edema (pericardially in extremities) and cardiac insufficiency manifested by tachycardia, dyspnea, edema, and no micturition or decreased circulation time, late at venous pressure and on specific ECG changes.

#### Treatment

- A Thiamine Hydrochloride U.S.P. Aqueous Hydrochloride B.P. 20-50 mg orally TID 1 M daily in divided doses for 2 weeks then 10 mg daily by mouth until deficiency improves.
- B Dried Yeast Tablets, U.S.P. (b.w.s.) 30 Gm tid
- C Diet Well balanced (2500-4500 Calorie) diet when tolerated

## VITAMIN B<sub>2</sub> (Rib-Flavin)

It is a functionally important coenzyme in the respiratory chain and is involved in the conversion of food to energy. It is readily absorbed from the intestine and is stored in the liver and in the kidneys. It is excreted in the urine. Its deficiency has been reported.

The recommended daily dietary allowances are as follows: Adults 1.7 mg (1700 µg); Infants 0.5 mg (500 µg); Pregnant women 2.0 mg (2000 µg); Lactating women 2.5 mg (2500 µg). The dietary sources include milk, eggs, meat, fish, and liver.

### ANTHRAQUINONE B<sub>2</sub> (Anthraquinone) (code No. 010 7622)

The etiological factors of anthraquinone deficiency are similar to those of thiamine deficiency. The intake of anthraquinone is important. The deficiency of anthraquinone is usually associated with thiamine deficiency but may occur separately.

A Mild or Early Manifestation (wasp-like superficial skin lesions, loss of appetite, and anorexia) and psychophobia (lack of interest in work and weight loss).

B Severe or Late Manifestation (Cheilosis (fissuring at the corners of the mouth), loss of the tip of the tongue, magenta tongue, and a mild or severe dermatitis, or a vascularization and hyperkeratosis in the skin or the bronchial dermatitis).

#### Treatment

A Mild or Early Manifestation L.S.P. Rib-Flavin B.P. 40-50 mg i.v. or

1 mg daily by mouth until all symptoms have cleared.

B Severe or Late Manifestation L.S.P. (or 10 mg yeast) 30 Gm. t.i.d.

C Well balanced (2500-4500 Calories) diet when tolerated.

## NIACTIN AND NIACTINAMIDE (P Factor)

This vitamin functions primarily in the CMO metabolism. Enzyme systems concerned with the drug transport and glycolysis. It is a component of the respiratory coenzyme NADH. A deficiency of 25-50 mg or more orally or i.v. causes cutaneous vasodilation with flushing, burning, itching, and sensations of warmth in 50% of persons. These symptoms are not produced by niacin itself.

The recommended daily dietary allowances are as follows: Adults 15 mg (1500 µg); Infants 5 mg (500 µg); Pregnant women 20 mg (2000 µg); Lactating women 25 mg (2500 µg). The dietary sources are liver, yeast, lean meat, whole grain cereal, peanuts, and potatoes.

### NIACTIN DEFICIENCY (Pellagra) (code No. 010 7623)

The etiological factors of deficiency are similar to those of thiamine deficiency. Niacin deficiency is the principal but not the only dietary defect in pellagra.

A Mild or Early Manifestation Multiple vague complaints and mild or moderate skin redness and hypertrophy of the papillae of the tongue.

B Severe or Late Manifestations Marked roughening of skin when exposed to light and friction; diarrhea, abdominal distention; early red tongue with atrophy of papillae; stomatitis; depression; clouding of mentality; rigidity and peculiar sucking reflexion.

#### Treatment

##### A Specific Measures

1. Nicotinamide U S P B P (Niacinamide) 50-500 mg 1 M I V or orally daily until all symptoms have subsided. Nicotinic Acid U S P B P (nicin) is less frequently used because of its vasodilating effect; dosage is similar.
2. Supplemental vitamins Give therapeutic doses of thiamin, riboflavin and pyridoxine (see dosage under each vitamin).
3. Dried Yeast Tablets U S P (beware of yeast) 30 cm tid.

##### B General Measures

1. Diet: Well balanced (2,500-4,500 Calories) high protein diet.
2. Symptomatic and supportive measures as indicated.
3. Dementia may require constant supervision.

#### Therapeutic Use of Nicotinamide in Cerebral Conditions

It may be used as a vasodilating agent for headache, myalgia, neurologic disorders and edema of the labyrinth (100 mg or more daily in divided doses). Nicotinamide does not possess this vasodilating effect.

### VITAMIN C (Ascorbic Acid)

Ascorbic acid participates in formation and maintenance of intercellular cement substance of all connective tissues (dermal cartilage, matrix of bone, collagen of fibrous tissue). It also participates in cellular metabolism of the tissue during growth. It is readily absorbed and excreted; the urinary excretion may be increased with formation of adrenocortical hormones. No toxicity has occurred in rat doses of 8 Gm daily.

The recommended daily dietary allowance are as follows: dose not (age 12-20 years) 75-100 mg; adult 75-75 mg; pregnancy 100 mg; lactation 150 mg. Therapeutic dietary sources are citrus fruits, tomatoes, peaches, bell peppers, and all green leafy vegetables. Copper use in heating, cooking and alkalinity all reduce vitamin C content of food.

### AVITAMINOSIS C (Scurvy) (code No. 010-763)

Avitaminosis C is usually due to inadequate intake, but may occur in a susceptible individual on a diet both quantitatively

A Mild or Early Manifestation Edema and hemorrhagic gingivitis; petechiae of the skin; and hypochromic anemia.

B Severe or Late Manifestation Severe muscle hemorrhages; swelling of the joints; rarefaction of bone; marked bleeding tendency; exostosis; blood in the sputum; malnutrition; or infection of the skin and pericardium.

C Treatment Daily oral C pill, read tannin reduced and the amount of leucocytes may show typical changes. The following of a uric acid blood level is a guide.

## Other Vitamins

### Treatment

#### A Deficiency

- 1 Sodium Ascorbate Injection 1 S.P., 0.5 to 1.0 Gm I.V. or I.M. daily in divided doses as long as deficiency exists
- 2 Ascorbic Acid U.S.P. B.P. orally in about the same doses

#### B Deficiency      Deficient      Ascorbic Acid, U.S.P. 200-300 mg per day orally

### Treatment of Miscellaneous Conditions

- A Vitamin C has also been used in the treatment of certain poisons in doses of 0.5 Gm or more. Proof of its value is lacking
- B Healing of wounds or ulcers or recovery from protracted diseases such as tuberculosis (dosage up to 300 mg daily orally)

## OTHER VITAMINS

### Pyridoxine Hydrochloride N.H.R.

May be important in the transamination and decarboxylation of proteins. Pyridoxine may relieve certain nervous symptoms and is also used in pellagra where niacin fails and may also relieve the glossitis and cheilosis in some persons unaided by riboflavin. Dosage is usually 10-50 mg I.V. or I.M. daily with other factors of the B complex.

### Choline

Choline is found in phospholipids and is a methyl donor, a lipotropic substance and a growth factor. It is found in large quantities in yeast. It has been used to prevent fatty changes in the liver parenchyma in chronic liver disease (see page 282).

### Folic Acid (Pt. reg. of ml. A 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, 35, 36, 37, 38, 39, 40, 41, 42, 43, 44, 45, 46, 47, 48, 49, 50, 51, 52, 53, 54, 55, 56, 57, 58, 59, 60, 61, 62, 63, 64, 65, 66, 67, 68, 69, 70, 71, 72, 73, 74, 75, 76, 77, 78, 79, 80, 81, 82, 83, 84, 85, 86, 87, 88, 89, 90, 91, 92, 93, 94, 95, 96, 97, 98, 99, 100, 101, 102, 103, 104, 105, 106, 107, 108, 109, 110, 111, 112, 113, 114, 115, 116, 117, 118, 119, 120, 121, 122, 123, 124, 125, 126, 127, 128, 129, 130, 131, 132, 133, 134, 135, 136, 137, 138, 139, 140, 141, 142, 143, 144, 145, 146, 147, 148, 149, 150, 151, 152, 153, 154, 155, 156, 157, 158, 159, 160, 161, 162, 163, 164, 165, 166, 167, 168, 169, 170, 171, 172, 173, 174, 175, 176, 177, 178, 179, 180, 181, 182, 183, 184, 185, 186, 187, 188, 189, 190, 191, 192, 193, 194, 195, 196, 197, 198, 199, 200, 201, 202, 203, 204, 205, 206, 207, 208, 209, 210, 211, 212, 213, 214, 215, 216, 217, 218, 219, 220, 221, 222, 223, 224, 225, 226, 227, 228, 229, 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1025, 1026, 1027, 1028, 1029, 1030, 1031, 1032, 1033, 1034, 1035, 1036, 1037, 1038, 1039, 1040, 1041, 1042, 1043, 1044, 1045, 1046, 1047, 1048, 1049, 1050, 1051, 1052, 1053, 1054, 1055, 1056, 1057, 1058, 1059, 1060, 1061, 1062, 1063, 1064, 1065, 1066, 1067, 1068, 1069, 1070, 1071, 1072, 1073, 1074, 1075, 1076, 1077, 1078, 1079, 1080, 1081, 1082, 1083, 1084, 1085, 1086, 1087, 1088, 1089, 1090, 1091, 1092, 1093, 1094, 1095, 1096, 1097, 1098, 1099, 1100, 1101, 1102, 1103, 1104, 1105, 1106, 1107, 1108, 1109, 1110, 1111, 1112, 1113, 1114, 1115, 1116, 1117, 1118, 1119, 1120, 1121, 1122, 1123, 1124, 1125, 1126, 1127, 1128, 1129, 1130, 1131, 1132, 1133, 1134, 1135, 1136, 1137, 1138, 1139, 1140, 1141, 1142, 1143, 1144, 1145, 1146, 1147, 1148, 1149, 1150, 1151, 1152, 1153, 1154, 1155, 1156, 1157, 1158, 1159, 1160, 1161, 1162, 1163, 1164, 1165, 1166, 1167, 1168, 1169, 1170, 1171, 1172, 1173, 1174, 1175, 1176, 1177, 1178, 1179, 1180, 1181, 1182, 1183, 1184, 1185, 1186, 1187, 1188, 1189, 1190, 1191, 1192, 1193, 1194, 1195, 1196, 1197, 1198, 1199, 1200, 1201, 1202, 1203, 1204, 1205, 1206, 1207, 1208, 1209, 1210, 1211, 1212, 1213, 1214, 1215, 1216, 1217, 1218, 1219, 1220, 1221, 1222, 1223, 1224, 1225, 1226, 1227, 1228, 1229, 1230, 1231, 1232, 1233, 1234, 1235, 1236, 1237, 1238, 1239, 1240, 1241, 1242, 1243, 1244, 1245, 1246, 1247, 1248, 1249, 1250, 1251, 1252, 1253, 1254, 1255, 1256, 1257, 1258, 1259, 1260, 1261, 1262, 1263, 1264, 1265, 1266, 1267, 1268, 1269, 1270, 1271, 1272, 1273, 1274, 1275, 1276, 1277, 1278, 1279, 1280, 1281, 1282, 1283, 1284, 1285, 1286, 1287, 1288, 1289, 1290, 1291, 1292, 1293, 1294, 1295, 1296, 1297, 1298, 1299, 1300, 1301, 1302, 1303, 1304, 1305, 1306, 1307, 1308, 1309, 1310, 1311, 1312, 1313, 1314, 1315, 1316, 1317, 1318, 1319, 1320, 1321, 1322, 1323, 1324, 1325, 1326, 1327, 1328, 1329, 1330, 1331, 1332, 1333, 1334, 1335, 1336, 1337, 1338, 1339, 1340, 1341, 1342, 1343, 1344, 1345, 1346, 1347, 1348, 1349, 1350, 1351, 1352, 1353, 1354, 1355, 1356, 1357, 1358, 1359, 1360, 1361, 1362, 1363, 1364, 1365, 1366, 1367, 1368, 1369, 1370, 1371, 1372, 1373, 1374, 1375, 1376, 1377, 1378, 1379, 1380, 1381, 1382, 1383, 1384, 1385, 1386, 1387, 1388, 1389, 1390, 1391, 1392, 1393, 1394, 1395, 1396, 1397, 1398, 1399, 1400, 1401, 1402, 1403, 1404, 1405, 1406, 1407, 1408, 1409, 1410, 1411, 1412, 1413, 1414, 1415, 1416, 1417, 1418, 1419, 1420, 1421, 1422, 1423, 1424, 1425, 1426, 1427, 1428, 1429, 1430, 1431, 1432, 1433, 1434, 1435, 1436, 1437, 1438, 1439, 1440, 1441, 1442, 1443, 1444, 1445, 1446, 1447, 1448, 1449, 1450, 1451, 1452, 1453, 1454, 1455, 1456, 1457, 1458, 1459, 1460, 1461, 1462, 1463, 1464, 1465, 1466, 1467, 1468, 1469, 1470, 1471, 1472, 1473, 1474, 1475, 1476, 1477, 1478, 1479, 1480, 1481, 1482, 1483, 1484, 1485, 1486, 1487, 1488, 1489, 1490, 1491, 1492, 1493, 1494, 1495, 1496, 1497, 1498, 1499, 1500, 1501, 1502, 1503, 1504, 1505, 1506, 1507, 1508, 1509, 1510, 1511, 1512, 1513, 1514, 1515, 1516, 1517, 1518, 1519, 1520, 1521, 1522, 1523, 1524, 1525, 1526, 1527, 1528, 1529, 1530, 1531, 1532, 1533, 1534, 1535, 1536, 1537, 1538, 1539, 1540, 1541, 1542, 1543, 1544, 1545, 1546, 1547, 1548, 1549, 1550, 1551, 1552, 1553, 1554, 1555, 1556, 1557, 1558, 1559, 1560, 1561, 1562, 1563, 1564, 1565, 1566, 1567, 1568, 1569, 1570, 1571, 1572, 1573, 1574, 1575, 1576, 1577, 1578, 1579, 1580, 1581, 1582, 1583, 1584, 1585, 1586, 1587, 1588, 1589, 1590, 1591, 1592, 1593, 1594, 1595, 1596, 1597, 1598, 1599, 1600, 1601, 1602, 1603, 1604, 1605, 1606, 1607, 1608, 1609, 1610, 1611, 1612, 1613, 1614, 1615, 1616, 1617, 1618, 1619, 1620, 1621, 1622, 1623, 1624, 1625, 1626, 1627, 1628, 1629, 1630, 1631, 1632, 1633, 1634, 1635, 1636, 1637, 1638, 1639, 1640, 1641, 1642, 1643, 1644, 1645, 1646, 1647, 1648, 1649, 1650, 1651, 1652, 1653, 1654, 1655, 1656, 1657, 1658, 1659, 1660, 1661, 1662, 1663, 1664, 1665, 1666, 1667, 1668, 1669, 1670, 1671, 1672, 1673, 1674, 1675, 1676, 1677, 1678, 1679, 1680, 1681, 1682, 1683, 1684, 1685, 1686, 1687, 1688, 1689, 1690, 1691, 1692, 1693, 1694, 1695, 1696, 1697, 1698, 1699, 1700, 1701, 1702, 1703, 1704, 1705, 1706, 1707, 1708, 1709, 1710, 1711, 1712, 1713, 1714, 1715, 1716, 1717, 1718, 1719, 1720, 1721, 1722, 1723, 1724, 1725, 1726, 1727, 1728, 1729, 1730, 1731, 1732, 1733, 1734, 1735, 1736, 1737, 1738, 1739, 1740, 1741, 1742, 1743, 1744, 1745, 1746, 1747, 1748, 1749, 1750, 1751, 1752, 1753, 1754, 1755, 1756, 1757, 1758, 1759, 1760, 1761, 1762, 1763, 1764, 1765, 1766, 1767, 1768, 1769, 1770, 1771, 1772, 1773, 1774, 1775, 1776, 1777, 1778, 1779, 1780, 1781, 1782, 1783, 1784, 1785, 1786, 1787, 1788, 1789, 1790, 1791, 1792, 1793, 1794, 1795, 1796, 1797, 1798, 1799, 1800, 1801, 1802, 1803, 1804, 1805, 1806, 1807, 1808, 1809, 1810, 1811, 1812, 1813, 1814, 1815, 1816, 1817, 1818, 1819, 1820, 1821, 1822, 1823, 1824, 1825, 1826, 1827, 1828, 1829, 1830, 1831, 1832, 1833, 1834, 1835, 1836, 1837, 1838, 1839, 1840, 1841, 1842, 1843, 1844, 1845, 1846, 1847, 1848, 1849, 1850, 1851, 1852, 1853, 1854, 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2021, 2022, 2023, 2024, 2025, 2026, 2027, 2028, 2029, 2030, 2031, 2032, 2033, 2034, 2035, 2036, 2037, 2038, 2039, 2040, 2041, 2042, 2043, 2044, 2045, 2046, 2047, 2048, 2049, 2050, 2051, 2052, 2053, 2054, 2055, 2056, 2057, 2058, 2059, 2060, 2061, 2062, 2063, 2064, 2065, 2066, 2067, 2068, 2069, 2070, 2071, 2072, 2073, 2074, 2075, 2076, 2077, 2078, 2079, 2080, 2081, 2082, 2083, 2084, 2085, 2086, 2087, 2088, 2089, 2090, 2091, 2092, 2093, 2094, 2095, 2096, 2097, 2098, 2099, 2100, 2101, 2102, 2103, 2104, 2105, 2106, 2107, 2108, 2109, 2110, 2111, 2112, 2113, 2114, 2115, 2116, 2117,

## Chapter 5

# DISEASES OF THE SKIN

## INTRODUCTION

### General Principles

#### A History and Examination

- 1 Take a careful history from every patient with skin disease
- 2 Do not neglect role of constitutional factors in production or aggravation of skin disease (Internal diseases nervous system factors diet hygiene etc.)

3 Examine entire body surface in good light

- #### B Physical Examination
- The first is a bewildering variety of dermatologic entities. It is best to be thoroughly familiar with the actions of a few and come to understand treatment methods that it is impossible to use a great many.

- 1 Consider the general character of the individual skin
  - a Dry skins usually require lubrication
  - b Moist oily skins usually require less drying
- 2 Begin treatment with mild simple medication
  - a Acute inflamed lesions require soothing or irritating
  - b Chronic thickened lesions require stimulating or keratolytic agents
- 3 Apply a preliminary amount of medication at a single small area to test skin sensitivity
- 4 Do not change remedies too frequently. Allow adequate time to act. However, discontinue remedy immediately in event of untoward reaction
- 5 In treating the patient as effectively on how to apply medication
- 6 When in doubt as to proper method of treatment UNDERTREAT rather than overtreat

## CORTICOTROPIN (ACTH) and CORTISONE

In continuation of the last chapter, we will now consider the use of corticosteroids. Corticotropin (ACTH) may be administered orally or by injection. This has been observed to be of benefit in the treatment of many types of skin disease. The most common use is in the treatment of allergic diseases and dermatoses. The use of corticosteroids in the treatment of skin disease is not uniform. Both during and after treatment, the patient and the physician may suppress or control the disease.

dermatoses but it does cure. It is important to understand the pharmacologic and pharmacologic effects of these drugs to achieve maximum benefits from therapy (see page 283).

The use of hydrocortisone ointment either in the acute or chronic form (1-2%) is very helpful in controlling pruritus associated with dermatitis and eczema.

## ANTIHISTAMINES

The antihistamine drugs form a group of chemically related agents which appear to block most of the characteristic effects of histamine. They do not block the release of histamine but evidently do prevent histamine from reacting on the end organs. In treatment they are used preventively upon the C<sub>4</sub>S but in higher doses they have an anesthetic effect. There is considerable variation in the tolerance and toxicity from drug to drug and from individual to individual. Comparative therapeutic doses of the various antihistamines have roughly similar toxic effects.

Therapeutic	Common	Uses	Antihistamine	Preparation
Drug	Regimental	to be used in	following	dosages
<b>A. Sedative Ethylamine Derivatives (NCH<sub>2</sub>)</b>				
1. Triphenylamine Hydrochloride	LSI	(Lyrizemine®)		60 mg
2. Methylamine Hydrochloride	44 R	(Thylone®)		250 mg
3. Thionylamine Hydrochloride	44 R	(Thylamine®)		250 mg
4. Lyrizemine	44 R	(Neo-antergan®)		50 mg
<b>B. Alkamine (NCH<sub>2</sub>) Diphenhydramine Hydrochloride</b>				
U.S.I. (Lyrizemine)				250 mg
<b>C. Miscellaneous Compound (CCH<sub>2</sub>N)</b>				
Chlorpheniramine				24 mg

### Indications

The antihistamine may be used effectively in nasal allergies, urticaria, angioneurotic edema, drug reactions, serum sickness, pruritus due to many forms of dermatitis. Menthol is a disease and motion sickness and are indicated to a lesser extent in other allergic states (e.g., allergic asthma and allergic migraine).

### Toxicity

The antihistamines may produce in some patients drowsiness, nervousness, dizziness, nausea, vomiting, diarrhea, muscular weakness, dryness of mouth, blurred vision, tremors and even at times convulsions.

## COMMON DISORDERS OF THE SKIN

## PRURITUS (Itching) (code No 143)

T ime 1

- A Sp e c i a l M a u e Remember that localized (as well as general) pruritus may result from systemic causes
- R move or treat specific causes whenever possible
  - 1 Skin infections (e.g. scabies, pinworms, pediculosis)
  - 2 Skin infections (e.g. fungal and bacterial infections)
  - 3 Skin inflammations (on infection) (e.g. lichen planus, eczema, urticaria)
  - 4 Allergic reactions (e.g. hyperhidrosis, anhidrosis)
  - 5 Allergic reactions (e.g. food, drug, clothing, serum, etc.)
  - 6 Sensitization (e.g. skin atrophy)
  - 7 Metabolic diseases (e.g. diabetes, hyperthyroidism, gout)
  - 8 Uremia
  - 9 Jund
  - 10 Opium intoxication (e.g. morphinism)
  - 11 Blood and neoplastic disease (e.g. leukemia, lymphoma)
  - 1 Pay homage to fact that (e.g. an elderly state)

B l o c k M a u e

- 1 Shave lotion, emulsions and ointments in incorporating the following analgesic and antipruritic ingredients on pages 100 and 107 may be of value in alleviating itching
  - 2 Relieve excessive dryness and moisten skin
    - a If skin is too dry, softening agents may afford relief (e.g. ointment (R 31 page 103))
    - b If skin is too moist, dryness may afford relief (e.g. with dry skin, oaks (R 18 page 98) shampoos (R 14 16 page 100) and powder (R 9 12 page 98) (especially if process is acute)
- Tub baths. Germinated potatoes may be effectively on the whole by lukewarm baths 15 minutes b.i.d. or t.i.d.
- All rubbing the skin should be blotted, not rubbed
- (1) Sodium bicarbonate baths 1 up sodium bicarbonate in tubful (50 gallon) of lukewarm water
  - (2) Starch and soda bath 1 3 p. M. and 1 up sodium bicarbonate dissolved thoroughly in tubful (50 gallon) of lukewarm water (Sod may be omitted)
  - (3) Starch bath 1 2 p. M. of or at night with effluvia to be taken to make an emulsion both, and then add to tubful (50 gallon) lukewarm water
  - (4) Tar bath 1 1 50 100 c. Solution of Coal Tar N.F. in tubful (50 gallon) of warm water (Wt. to be as follows)

**CAUTION** Avoid excessive drying of skin by over-bathing prolonged bathing period and exposure to drafts after bathing

C G e n e r a l M a u e

- 1 Diet
  - Foods should be simple. Avoid rich and spicy food
  - 2 T. diets or limit the diet should be used in up to 100 of 100 (see page 56)
- 2 Pay heed to pruritus is particularly common



## 88 Contact Dermatitis

an anti ty state obsession, compulsion, or a psychotic disorder direct therapy accordingly

- 3 External irritants (e g rough clothing occupational contactants) should be avoided
- 4 Bathing practices Soap should be avoided in individuals with dry or irritated skin Starch bath may be used (see previous page)
- 5 Nails should be kept trimmed and cleaned
- 6 Avoid scratching, if possible because of vicious cycle which can be established
- 7 Unnecessary medication should be stopped since medication itself can often produce pruritus
- 8 Antipruritic drugs The following agents may be of benefit
  - a Calcium salts 10 cc of 10% calcium gluconate i v slowly once daily or every other day p r n
  - b Antihistaminic drugs may be tried in certain cases of pruritus of allergic or undetermined etiology For a list of commonly used antihistaminic preparations see page 88
  - c Epinephrine 0.25 to 0.5 cc (4 to 8 min) of 1:1,000 solution every 4 hours may be of value in acute cases suspected of being due to allergy (urticaria)
  - d Phenobarbital, 0.013 to 0.03 Gm (1/4 to 1/2 gr) b i d or q i d may provide useful sedation in agitated or emotionally distressed patients Remember that barbiturates in themselves may produce dermatitis (rarely)
  - e Autohemotherapy Some dermatologists advise the injection of 10 cc of the patient's whole venous blood into the hip muscles every 48 hours for 3 injections
  - f ACTH gel 10 to 40 mg i m once or twice weekly or cortisone 25 to 100 mg daily by mouth

### DERMATITIS VENERATA (Contact Dermatitis)

(code No 110-3001)

(Dermatitis Venerata Due to Plant Irritants code No 110-378)

An acute or chronic dermatitis which results from direct contact of chemicals or other irritants with the skin Lesions are most often on exposed parts and may be asymmetric (cf due to internal agents) Lesions are aggravated by exposure to the irritant and this should be avoided Patch tests may be of value in diagnosis as corroboration of clinical impressions

#### Diagnosis

Survey the patient's environment and study his total activities to determine irritant

- A Search for a history of recent exposure to new chemicals drugs soaps cosmetics or other contact irritants The location of the lesions may be of value in identifying the irritant e g scalp (rinses or shampoos) face (soaps shaving materials cosmetics) neck (jewelry clothing) trunk (clothing) upper extremities (soaps cosmetics plant toxins industrial chemicals) and lower extremities (stockings shoes shoe dyes)

- B Use protective isolation in certain selected cases c. ution re exposure may help to establish the irritant
- C Patch tests may be of value but false positive and false negative reactions may occur Dermatitis produced by such tests should resemble the clinical dermatitis In the event of a positive reaction, a control test should be done on a normal individual

### Treatment

#### A. Definitive Measures

- 1 Prevent re-exposure to irritant
  - a Avoid soaps and detergents
  - b Cosmetics Change to so-called non-allergic nontoxic or eliminate cosmetics entirely
  - c Occupational irritants
    - (1) Protective rubber gloves may be used but a good indicator in such cases an inner cotton glove must be used
    - (2) Protective creams (barrier creams) may be tried but are almost useless
    - (3) Change of occupation or duties to those not involving use of irritant agents may be necessary
  - d Plant irritants (especially Rhus species e.g. poison ivy)
    - (1) Detection of plant by means of removal by hemi-solamine (2,4-D or dihydroxyacetic acid) as dwellings and in areas frequented by people
    - (2) Avoidance of Rhus infestation
- 2 Prompt and thorough removal of irritants by prolonged washing or by removal with solvents or other chemical agents may be effective if applied very shortly after exposure In the case of Rhus toxin thorough washing with soap and water must be done within a few minutes if it is to be of any value

#### B. Local Management Treatment stage and type of dermatitis (B)

Pg 95 97 100)

- 1 Acute weeping dermatitis
  - a Do not scrub lesion with soap and water
  - b Apply soothing solutions (table on pag 98) If upstention be more generally with soothing starch and boric acid antipruritic bath mentioned on pag 87 Shaking lotion (R 14 15 pag 100) may be indicated that all of wet dressing or in ite also between wet dressings especially in it trigonous areas where cooling is not marked Lesion on the extremities particularly may be bandaged with wet dressings
  - Hydrocortisone ointment 1% 2% applied sparingly 2-4 times daily may be very helpful
- 2 Subacute dermatitis (subiding) Use bak lotion
- 3 Chronic dermatitis (dry and lichenified) Treat with hydrophilic or oily ointments or creams Treatments perhaps most useful in this stage of the dermatitis

#### C. General Measures

- 1 Antihistaminic drug orally may be of use (see pag 66)
- 2 ACTH gel, 20-40 mg IM or cortisone 25-100 mg orally (with despredsone) may be tried and perhaps daily (S pag 423)

## ERYTHEMA NODOSUM (Due to Infection code No 114 1x0)

A tender nodular erythematous dermatosis occurring most commonly on the extensor surfaces of the legs and (less often) forearms. It is usually caused by toxins of infections and occasionally by drugs. The disease occurs most commonly in the spring or fall and usually runs a course of 2-6 weeks or longer.

### Treatment

#### A General Measures

- 1 Eliminate or treat the specific cause
  - a Infections. Almost all infections, bacterial, tuberculous, mycotic or viral, are capable of causing erythema nodosum. For treatment see specific diseases.
  - b Exogenous toxins. E.g. drugs or chemicals.
- 2 Rest. Hospitalization may be advisable.
- 3 Focal infections. May be corrected although this does not appear to influence the course of the disease.

#### B Local Measures. Usually unnecessary but if lesions are troublesome or complicated, treat according to stage and type of dermatitis (see pages 96-97 and 108).

#### C Terramycin® or Aureomycin®, 250 mg q.i.d. for several days may be useful.

## ERYTHEMA MULTIFORME

(Infection code No 114 190) (Poison code No 114 3x7)

An acute inflammatory polymorphic skin disease of multiple or undetermined origin. There is often a history of drug exposure or of recent or current infection. The skin lesions are found most frequently on the dorsa of the hands and forearms and on the feet, neck, cheeks, buccal mucous membranes and genitalia. The illness is usually self-limited although it is frequently recurrent.

### Treatment

#### A General Measures

- 1 Eliminate causative factors
  - a Chronic systemic infections (e.g. tuberculosis)
  - b Focal infections
  - c Sensitizing drugs
- 2 Penicillin parenteral, 150,000-300,000 units i.m. is said to decrease the extent and duration of the illness when a secondary infection is present.
- 3 Terramycin® or Aureomycin®, 250 mg q.i.d. for several days may be useful.

#### B Local Measures

- 1 Mild rest and good nursing care when fever is present.
- 2 Antihistaminic drugs may be tried but results are equivocal. For a partial list of official and accepted preparations see page 88.

#### C Local Measures. Treat stage and type of dermatitis (see pages 96-97 and 108).

- 1 Acute lesions. Employ simple wet dressings and soaks or soothing lotions. For treatment of bullous lesions see page 261.

## 2 S bacute lesions - Soothing lotions

Prophylaxi

Avoid il unnecessary m dication in su c ptible individu la  
 i e p tle ts with previous history of ythema multiform

**ECZEMA (code No 111 390)(and Eczematoid Dermatitis)**

A l rg group of non specific acute or chronic supe ficial in  
 flamm tory skin reactions which occur as result of exposur t  
 ch mical physiol or unknown irritant or as a result of M rge s  
 irritants may be ext rn l (e g c tact d rmatiti ) o intern l  
 ( g d m titis m dic mentosa) The e m y be a histo y f  
 allergic tende le (topl ex ma) and blood eosinophilia may be  
 found Th t rm ec ematoid d rm titi i u d for ecsem lik  
 e ctions of undetermined o igin Th l sions of e em a usually  
 pruritic Acut lesions are usually ryth matus vesicular o  
 exudativ Chronic le ons are usually thicken d de quematit e  
 li h nified

Tre tm tA Sp ifi M

- 1 Eliminat ion of itching ag nt ( b vel) m in a s s th  
 only spe ific m asure Ac reful history tri l and e r  
 lmin tation and exposure technic may b of val in in  
 lmin ting sp ifi off ding g t Skin t st ar ften  
 v lu less De nettition i f no val e Sen itivities are  
 us lly m itip)
- 2 Diet Sho ld b ad q t and w ll b lan d Th l no  
 vid t gg t th t standard ed outline diet ry r  
 at lction ar of l e esp cially in ad lts Trial d t o  
 lliminati n diets m y be of lu in d t rmining food ller  
 g s in individual ca s wh an urti il component is  
 prese t F od d i l m y be k pt by pati nts with h onl e  
 m to d t rmine possibility of food ll gy Reported  
 common food offend ra a wh t milk gg po k fish  
 sh llf h tom toes m awb i and hocol te
- 3 Pay hoth py A att mpt m y be m de to determin and  
 c t e i ting emoti l d t u bances b t this is of no  
 pr ctical valn
- 4 R mo d flint fo i of infecti n B t a m outin poly  
 urge y

B Ge e l M asu e

- 1 Antibi t minal drugs occasionally p o lde b neficial r sults  
 although spons in gen ral is dis ou ging (see p ge 68)
- 2 ACTH o ti on may provid spect ular imp o me t in  
 sev or fulbrant ma ( p g 423)

C Loc l T tm t

- 1 A id il unnn ary local ir R tion to the kin su h  
 may o ur f om e siv b thing o s a r sult of xpo ur  
 to irrit ting drugs hemi al g e and so p S pl  
 det g nts a not advis bl Cl ar up skin inf ctions  
 p omptly (particula ly tho with xps tes) by approp lat  
 m asur ( e p ges 85 87)

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- 2 Hydrocortisone 1% ointment applied sparingly twice a day may be very helpful
- 3 Treat the clinical type and stage of the dermatitis
  - a Acute weeping lesions Use solutions listed in table on page 88 as soothing or astringent soaks baths or wet dressings in the daytime for 30 minutes t i d or q i d Shake lotions (§ 14 15 page 100) may be employed at night or when wet dressings are not desirable Lesions on extremities particularly may be bandaged for protection at night Powders (§ 9 11 12 page 89) may be used in intertriginous areas when oozing is not marked
  - b Subacute or subsiding lesions may be treated with shake lotions which may incorporate mild antipruritic or mild stimulating agents (see page 107) Shake lotions are usually preferred for widespread lesions Ointments (see page 104) containing mild tar may be used (see table on page 106)
  - c Chronic dry lichenified lesions are best treated with ointments creams and pastes (see page 107 3) employing lubricating keratolytic antipruritic and mild keratoplastic agents mentioned in the table on pages 106 7 as indicated The tars are perhaps the most popular therapeutic agents in chronic eczema (§ 5% coal tar in ointments creams and pastes)
- 4 X ray therapy may be used effectively if only temporarily in many stages Treatment by x rays must be reserved for the specialist

### DERMATITIS MEDICAMENTOSA ( Drug Rash ) (code No 110 3 )

An acute or chronic inflammatory skin reaction which is caused by a wide variety of drugs and which causes a wide variety of skin lesions in susceptible individuals The reaction may be immediate or delayed (to a few weeks) and may or may not be associated with constitutional disturbances (fever headache etc) Improvement following withdrawal and elimination of the suspected drug usually takes a few days but may take longer As a rule it is not advisable to attempt a diagnostic provocation or an elimination by re exposure to the drug Skin tests are seldom of any value

#### Treatment.

##### A Specific Measures

- 1 Stop all drug if possible
- 2 Hasten elimination of drug by increasing fluid intake
- 3 Give specific detoxifying agents
  - a Dimercaprol, U S P (BAL<sup>®</sup>) may be tried in cases due to heavy metals (arsenic mercury gold etc) (see page 536)
  - b Sodium chloride 5 to 10 Gm (75 150 gr) daily orally may hasten elimination of bromides and iodides in cases due to those drugs (see page 538)

##### B General Measures.

- 1 Discontinue all unnecessary medication, when feasible for

as long as possible as possible

- 2 Treat systemic manifestation as they arise e.g. an mictic icterus purpura etc
- 3 A thistaminic may be of value in treatment of urticarial and angioneurotic hives (see page 66)

See 1 M 2 Treat the various stages of dermatitis according to the major dermatitis which is simulated

- 1 Eczematoid (see page 71)
- 2 Allergic (see page 78)
- 3 Pruritic (see page 67)
- 4 Pyoderma (see page 85)
- 5 Urticarial (see page 78)
- 6 Bullous (see page 98)
- 7 Lichenoid (see page 74)
- 8 Exfoliative (see below)

### EXFOLIATIVE DERMATITIS (code No 110 968)

A toxic cutaneous reaction often due to sensitization to certain drugs (e.g. arsenic and gold) but more commonly used by lymphoblastoma. It is characterized by itching weeping erythematous patches which rapidly cleave and spread to become generalised. Finally a desquamation or exfoliation of large areas of skin occurs. There is an associated severe constitutional reaction with fever and other systemic symptoms. The disease runs its course of weeks to months and is attended with a high mortality rate.

Treatment This is a medical emergency

A Stop All Medication

- 1 Stop all drugs if possible
- 2 Hasten elimination of offending drug by all means e.g. by inducing fluid diuresis
- 3 Dimerpoxol USP (BAL<sup>®</sup>) This drug may lessen the severity of the reaction due to arsenic and gold (see page 838)
- 4 ACTH 20-40 mg I.V. or I.M. or cortisone 50-100 mg I.V. or by mouth daily if indicated

B General Measures

- 1 Patient admitted to hospital when possible. Maintain on bed rest
- 2 Keep room temperature constant throughout. Avoid drafts
- 3 Institute appropriate measures transfusions plasma etc indicated
- 4 Avoid all unnecessary medication
- 5 ACTH or cortisone may provide symptomatic improvement in severe fulminant exfoliative dermatitis (see page 423)
- 6 Secondary infections. Penicillin or other antibiotic drug should be given when the evidence of bacterial infection (see pages 85-87 for dosage schedule). Pyoderma is the most common complication of exfoliative dermatitis

C Local Measures

- 1 Observe skin hygiene
- 2 Avoid irritating local applications
- 3 Treat skin surface with emollient ointments  
First Wet dressings soothing bath (see page 87)  
Emollient (see page 99) and haemolysis (see page 100)  
b. Late in the course of the disease (see page 100) and intermittent (see pages 102-103)

- 4 Topical anti-infective drugs (e.g. 1% aqueous neomycin, Terramycin® Aureomycin® chloramphenicol erythromycin, or polymyxin B ointments) should be used very cautiously and only when necessary (see page 85-87 and 107)

### Precautions

Patients who are receiving drugs capable of producing severe allergic dermatitis should be watched carefully for development of skin reactions of all types while under therapy. The drug should be withheld until the nature of any skin reaction is determined. Definite sensitization may be considered an absolute contraindication to further drug administration.

## DERMATITIS ACTINICA (code No 110-451) (Erythema Solare or Sunburn)

An acute inflammatory skin reaction following exposure to solar or other ultraviolet radiation. It may vary from simple erythema to severe exfoliation and may be associated with systemic manifestations. Some individuals are abnormally light sensitive.

### Treatment

- A Symptoms is mild to moderate. Treat constitutional symptoms by appropriate supportive measures. Control pain, burning, fever and gastrointestinal and other symptoms as they arise.
- B Local is mild. Treat as for any acute dermatitis (see page 108). First use cooling and soothing wet dressings (see page 98) and follow with lotions (see page 100). Greases should be avoided because of their occlusive effect.

### Precautions

- A Individuals with very blond sensitive skins should avoid strong and prolonged exposure to the sun or ultraviolet radiation. Preliminary conditioning by graded exposure is advisable.
- B Protective Agent. Apply to skin before exposure to radiation.
- 1 Para-aminobenzoic acid 10% in hydrophilic ointment
  - 2 Carbolsol (phenolized) Vaseline® is a good sunscreen
  - 3 Menthyl anthranilate (5%) and 5% titanium dioxide cream
  - 4 Diglycidyl ether of bisphenol A (DGEBA)

## LICHEN PLANUS (code No 110-965)

A chronic inflammatory skin disease of unknown cause characterized by small flat topped violaceous pruritic papules which are angular in shape (usually quadrilateral) and of varying sizes. They occur commonly on the flexor surface of the forearms and inner thighs on the lower part of the back and on the genitalia. There may be associated buccal lesions. Histological pigmentation and atrophy may occur but usually there are no sequelae. Lichen planus may be associated with drug eruptions (hydralazine, quinacrine).

### Treatment

- A Constitutional is mild.

- 1 Phenobarbital 0.015 to 0.03 Gm ( $\frac{1}{4}$  to  $\frac{1}{2}$  gr) b.i.d. q.i.d.
- 2 Psychotherapy Patients are often high strung o.t. and nervous. Episodes of dermatitis may follow emotion. Mental status should be directed to relieving anxiety.
- 3 Bismuth subsalicylate 0.2 Gm (3 gr) i.M. once weekly for a total of 6 to 12 or more injections has been suggested.

**B Local Measures**

- 1 Make lotion containing tar (§ 17 page 100)
- 2 X-ray may be used only in cases with thick plaques. If difficulty to other forms of treatment. Treatment by x-ray must be reserved for the psoriasis.

**PSORIASIS (code No. 111.961)**

Acut or chronic inflammatory skin disease. It is characterized by multiple and papulopustular lesions of varying size and configuration (usually with well defined borders). The lesions have dry livid color and bleeding occurs when the scales are removed. Pruritus is a frequent accompaniment. Rupture of vesicles. The lesions occur on the extensor surfaces of the extremities and on the trunk and scalp. There is sometimes an associated dermatitis but no constitutional effect. Stippling of the skin may be pathognomonic.

**Treatment**

**A General Measures**

- 1 Climate. Warm climate seems to exert favorable effect.
- 2 Nasopharyngeal internal medication (postoperative lesion) of the nasal cavity with the potassium permanganate solution. The dose is 1 to 2 drops in water 3 to 4 times a day of the lesions and the dried effect of the solution of resins (K<sub>2</sub>Cr<sub>2</sub>O<sub>7</sub> solution).
- 3 Vitamin D 100,000 to 1,000,000 units daily has been recommended. Results equivocal.
- 4 A 1% solution of salicylic acid (potassium permanganate solution) has been recommended in the treatment of the disease. Do not use for the administration and the use and venodilation of the drug is objectionable. It may be given in repeated courses if indicated by the course of the disease. The greater than 2 to 3 months (page 238).
- 5 Crude extract 1 to 2 c.c. i.M. 2 to 3 times a week. Results equivocal.
- 6 Psychotherapy. An important factor in the treatment of the disease. The goal is the relief of the disease. An attempt should be made to relieve the patient's anxiety.

**B Local Measures**

- 1 At psoriasis (avoid irritation, stimulation, drying). Begin with a make lotion (§ 14 to 15 page 100) or blinding ointment (§ page 102) containing 5% of the tincture of coal tar.
- 2 A 1% solution of salicylic acid is gradually incorporated into the



- a keratoplastic agents (see page 106) into lotions (see page 100) and hydrophilic ointments (see page 103). Watch patient carefully
- 2 Subacute psoriasis
- a Give warm baths daily scrubbing the skin lesions thoroughly with brush soap and water
  - b Apply increasing concentrations of keratoplastic or sloughing agents (see pages 106 and 107) incorporated in lotions (see page 100) and hydrophilic ointments (see page 103)
  - c Solar or ultraviolet irradiations may be applied in gradually increasing doses
- 3 Chronic psoriasis
- a Ammoniated mercury ointment 3% locally b i d
  - b Anthratin ointment 4% locally once a day (a old ey ill)
  - c Combined ultraviolet irradiation and tar regimen (modified from Coe's regimen). To be carried out daily as needed
    - (1) Smear 2-5% coal tar ointment (see page 72) thickly on skin and allow to remain for 12-24 hours
    - (2) Wipe off ointment with mineral oil leaving light stain
    - (3) Follow with daily graded suberythema doses of ultraviolet light as tolerated

### PITYRIASIS ROSEA (code No 111 962)

A common, mild acute inflammatory skin disease of unknown etiology which is characterized by a papulosquamous eruption on the trunk arms and thighs and which occurs more frequently in the spring and fall. The papules are pink and oval with scaling borders and pale centers. They are typically arranged with their long axes along the cleavage lines of the skin. A single herald patch may precede multiple lesions by a period of several days. The lesions may or may not be pruritic. The disease usually lasts 8 weeks with or without treatment.

#### Treatment

A General Measures None

B Local Measures

- 1 Acute irritated lesions are uncommon. If present treat as for acute dermatitis with wet dressings (see pages 88 and 92) or with alkali lotions (see page 100 101)
- 2 Detergent solution of coal tar 5% in starch lotion b i d
- 3 Ultraviolet light is helpful
- 4 Pruritus See local antipruritic measures on page 67

### SEBORRHEIC DERMATITIS (code No 111 190)

An acute or chronic papulosquamous dermatitis often associated with excessive oiliness of the skin and occurring in the so-called sebaceous areas of the body (scalp midportion of face sternal region and intertriginous areas). The lesions appear (1) as yellowish greasy scales or (2) as an acute or chronic erythematous dermatitis in areas of sebaceous gland concentration and (3) intertriginous areas. Lesions frequently are pruritic.

TreatmentA General 1 M

- 1 Diet Well balanced adequate diet avoiding excess sweets, pices, hot drinks and alcoholic beverages
- 2 Regular working hours, recreation and sleep
- 3 Simple leanliners
- 4 Remove aggravating systemic factors (infections, overwork, emotional stress, constipation and dietary abnormalities)

B Local 1 M Treat type and stage of dermatitis

- 1 Acute, subacute or chronic eczematous lesions Treat locally as for dermatitis or eczema (see page 71)
- 2 Seborrhea of scalp
  - a Carbolic shampoo 1-2 times a week with mild soap
  - b Ammoniated alum 5% or a lotion of coltar 5% employed in hydrophilic ointment base (see page 103) can be rubbed well into scalp 1-3 times a week and followed by shampoo
  - c Mild coal tar scalp lotion (R 31, page 101) may be used
- 3 Seborrhea of the hairy area Mild stimulating lotion (R 17, page 100 or 20, page 101) may be used. Ointment (R 36, page 104) or 3-5% in hydrophilic ointment (see page 103) may be used. (The addition of 1% salicylic acid is more general)
- 4 Seborrhea of intertriginous areas Avoid greasy ointments. Astringent wet dressings (R 16, page 98) followed by 5% ammoniated alum scrub in hydrophilic ointment (see page 103) may be used.

**EXTERNAL OTITIS (code No. x75 100)**

This may be considered a variant of seborrheic dermatitis and often may be complicated by infection. Eczematoid dermatitis. A interlaced with crusting or thickening due to inflammation of the canal wall and predisposition to secondary bacterial infection usually with *Pseudomonas aeruginosa* (*Bacillus pyocyaneus*). Fungi are rarely if ever associated.

TreatmentA General 1 M

- 1 Penicillin, 300,000 units once or twice daily 1 M for the accompanying fever and erythematous changes
- 2 Phenobarbital, 0.015 to 0.03 Gm ( $\frac{1}{4}$  to  $\frac{1}{2}$  gr) b i d q i d

B Local 1 M Treat

- 1 Acute lesions Cool wet dressings
- 2 To remove ceruminous debris if present Glycerine of hydrogen peroxide with carbamide as erasers b i d
- 3 3% Vioform® or amiodoly b i d
- 4 Hydrocortisone ointment 1-2 1/2% loc b i b i d
- 5 X-ray therapy in refractory cases (must be given only by a trained specialist)
- 6 Polymyxin B bacitracin ointment (Poly-Bact®)  
Tetracycline® A reomylin® neomycin® or erythromycin® ointments (see page 14)

## ACNE VULGARIS (code No 151 Tx0)

A common inflammatory skin disease of genetic origin provoked by androgens in the male and progesterone in the female. It is usually found in adolescents with pleomorphic lesions (pustules, blackheads, whiteheads, enlarged pores, cysts and scarring) localized typically on the face, neck, chest, back and shoulders.

TreatmentA General Management

- 1 Diet should be adequate and well balanced. Avoid excess of carbohydrates, chocolate, nuts, fatty or fried foods, alcoholic beverages, and spicy foods.
- 2 Eliminate all unnecessary medication, especially bromides or iodides.
- 3 Avoid occupational exposure to mineral oils and greases.
- 4 Endocrine perturbations. Estrogens may be tried in the female.
  - a Estrolic coated stilbestrol 0.3 to 1.0 mg (1/20 to 1/10 gr.) daily by mouth.
  - b Estrogenic substances containing (1 cm<sup>2</sup> ring) 1.2 mg (1/40 gr.) daily or
  - c Piperazine citron sulfate (Sulestrin<sup>®</sup>) 1.5 mg (1/40 gr.) daily.
 Estrogens should be stopped for one week (or menstrually) each month. They should not be used if there is a history of breast or genital malignancy or if chronic cystic mastitis. A periodic Papanicolaou smear of the uterine cervix and vagina is recommended.
- 5 Aquasol vitamin A 100,000 units orally each day for 3 months may be tried but has limited value.
- 6 Correct systemic derangements. Indigestion, constipation, malnutrition, infection, anemia, and emotional disturbances.
- 7 Vaccines. Autogenous and stock vaccines and other foreign protein antigens have been employed with equivocal results.

B Local Management

- 1 Local cleansing of skin and scalp.
  - a Ordinary soap for cleansing.
  - b Avoid greasy cleansing creams and other cosmetics.
  - c Shampoo scalp 1-2 times a week (see page 103).
- 2 Extraction and drainage of local lesion. In selected cases only.
  - a Extract blackheads with comedo extractor after softening face with hot water compresses for 1/2-1 hour.
  - b Incise and drain fluctuant cystic lesions with small sharp scalpel. Hot compresses 1/2 hour tid favor drainage.
- 3 Keratoplastic and keratolytic agents.
  - a Hot water or boric acid compresses (not steaming) may be used to produce hyperemia and desquamation of lesions.
  - b Keratolytic lotions. Acne lotion (see if retinol lotion) (see page 101) or sulfur resorcinol lotion (see page 101) may be tried. They are applied locally to the skin at bedtime and washed off in the morning.
  - c Keratolytic ointments and pastes. Begin with weak preparations and build up a tolerated. Apply at bedtime and

- r move in the morning  
 (1) Sulfur 2 10% in hydrophilic ointment (s pag 103)  
 (2) Sulfur and kaolin paste (R 39 p 104)  
 (3) Quinolor<sup>®</sup> ointment or Vioform<sup>®</sup> ointment (s pag 107)  
 4 I radiation  
 a Simple exposure to sunlight in graduated doses  
 b Lotion: 1% May be used as an adjunct to other treatment or to remove scurf. Use as by the manufacturer. This is up to point of mild erythema and X-ray. This is effective in the treatment of severe allergic reactions for only the most severe cases. Do not use other measures concurrently. List

# URTICARIA (Hives) (code No 11x 390) and ANGIONEUROTIC EDEMA (Giant Hives) (code No 11x 380)

An acute or chronic inflammatory skin condition characterized by multiple raised, red, itchy wheals. The wheals are usually transient, lasting from a few minutes to a few hours, but may persist for days. The condition is often associated with allergic reactions, but may also be caused by other factors such as infection, drugs, or physical factors. The treatment is symptomatic, and may include antihistamines, corticosteroids, and other measures to relieve the itching and inflammation.

- Treatment:
1. Purgation: Initial purgation to remove possible antigenic material has been recommended. (1/2 to 1) r i m i  
 0.065 to 0.13 Gm (1/2 to 1) may be given. Purgative stool may be used for initial purgation.
  2. Diet: During the acute phase, a strict diet should be followed. Avoidance of all foods which may cause allergic reactions is advised. This includes avoidance of all animal products, especially milk and eggs, and avoidance of all fruits and vegetables. A strict diet should be followed until the condition has subsided. The diet should be liberalized gradually as the condition improves.
  3. Antihistamines: Antihistamines are the mainstay of treatment. They should be given in full therapeutic doses. Examples include: Chlorpheniramine, Clemastine, and others.
  4. Corticosteroids: Corticosteroids may be used in severe cases to suppress the allergic reaction. They should be given in full therapeutic doses for a short period of time.
  5. Other measures: Other measures to relieve the itching and inflammation include: cool compresses, calamine lotion, and topical corticosteroids.

## 80 Intertrigo

(2) Urticaria is intense

(3) Antihistaminic drugs have failed to give relief

c Ephedrine sulfate 0.025 Gm (3/8 gr) orally q.i.d.

d Ephedrine Sedative mixtures for therapy or prophylaxis

(1) Ephedrine sulfate and pentobarbital sodium

℞ Ephedrine sulfate 0.025 gr 3/8

Pentobarbital sodium 0.025 gr 3/8

Sig One q.i.d.

(2) Ephedrine sulfate and phenobarbital

℞ Ephedrine sulfate 0.025 gr 3/8

Phenobarbital 0.015 gr 1/4

Sig One q.i.d.

5 ACTH or cortisone may provide spectacular improvement in severe or relapsant angioneurotic edema (see page 423)

These drugs should be used only if it is apparent that the patient will not respond to more conservative measures

6 Miscellaneous measures have been recommended for the chronic forms of the disease but their value is questioned

a Hydrochloric acid dilute 15 8 gr t.i.d. a.c. and during meals Brush teeth after meals with sodium bicarbonate

b Calcium gluconate 1.0 Gm (15 gr) t.i.d. orally p.c.  
Other calcium salts may also be used

B Local Measures A tipuristics are frequently of benefit

1 Soothing antipruritic baths (see page 67)

2 Soothing antipruritic lotions (see page 100)

### Prophylaxis

A Eliminate and avoid re-exposure to causative factors

1 Sensitizing drugs Almost all drugs are capable of producing an urticarial reaction. Opiates barbiturates salicylates penicillin sulfonamides bromides iodides antihistaminics ACTH etc

2 Sensitizing foods Any food may produce an urticarial response in susceptible individuals and should be considered in obscure cases (particularly chronic cases) of urticaria

3 Aggravating physical factors e.g. excess heat and cold skin and mucous membrane irritants

4 Aggravating systemic factors e.g. chronic infections foci of infections parasitic infestations and blood dyscrasias

B Relief of Psychic Disturbance In susceptible individuals emotional stress and strain may precipitate the lesions

## INTERTRIGO (code No 111-437)

Erythema due to chafing of the skin

### Treatment

Treat as tinea cruris but do not use fungicidal agents (see page 90)

# MILIARIA (Heat Rash) (code No 153-445)

81

An acute dermatitis characterized by small erythematous burning, and often pruritic papules, vesicles and pustules which occur most commonly on the upper extremities, trunk and intertriginous areas. It is caused by exposure to a hot moist environment.

Treatment

## A General Measures

1. Provide optimal working conditions when possible i.e. controlled temperature, ventilation and humidity.
2. Avoid wet bathing and use of strong irritating soaps.

## B Local Measures

1. Antipruritic cooling lotion apply b.i.d. to q.i.d.
 

Menthol	10	gr. av
Phenol	30	gr.
Glycerin	150	
Alcohol 35% q.s. ad	340	0
2. Drying astringent lotion (H 16 with 1% phenol) (H 15 page 100)
3. Sulfur resorcinol lotion (for seborrheic skin) (H 20 page 101)
4. Antipruritic powder or other dusting powders (see page 98)
5. Treat secondary infections (pyoderma, superficial) with potassium permanganate soaks compresses or baths (see page 98). Ammoniated mercury 2.5% in a hydrophilic ointment (see page 103) may be employed advantageously.
6. Tannic acid 10% in 70% alcohol locally b.i.d.

## Prophylaxis

- A. Toughen or tan skin. Graduated exposure (increased daily) to sunlight or ultraviolet light for individuals who will later be subjected to hot moist atmosphere.
- B. General Measures (see above).
- C. Avoid exposure to adverse atmospheric conditions for extremely susceptible individuals.

## AND-GENITAL PRURITUS

(Anal code No 143-573) (Vulva code No 771-570)

Differential

- A. Consider the effect of systemic causes of pruritus, anxiety etc. and diabetes mellitus and intestinal parasites.
- B. Rule out all obvious local pathological conditions of the anus and rectum, bowel irregularities or local cutaneous diseases.

Treatment (See also Pruritus page 67)

## A General Measures

1. Diet. Avoid hot spicy foods (e.g. hot peppers or chili) and drugs which can irritate the anal mucosa.
2. Treat constipation if present (see page 254).
3. Provide necessary proctologic treatment as indicated.
4. Instruct the patient to use very soft or moistened toilet cloth after a bowel movement and to clean thoroughly.
5. Warn patient should apply appropriate cautions for use.
6. Instruct the patient regarding harmful and poisonous.

## 82 Callosities

### *It is of scratching*

#### B Local Measures

- 1 Calamine lotion with 1% phenol applied locally
- 2 Sitz baths b i d if the area is acutely inflamed and oozing, using 1 IB 000 1 200 (0.01-0.5%) silver nitrate 1 IB 000 (0.01%) potassium permanganate or 1 IB (5%) aluminum acetate solution
- 3 Bandaging should be changed daily
- 4 Control excessive perspiration by use of drying powders such as talc (see page 93)
- 5 Paint fissured or ulcerated areas with 3-10% silver nitrate
- 6 1-2% hydrocortisone ointment locally b i d
- 7 X-ray therapy may be used if other measures fail. This should be reserved for the specialist

#### Prophylaxis

- A Treat all possible systemic or local causes
- B Instruct the patient in proper and general hygiene

### **CALLOSITIES (code No. 112-410) and CORNS (of feet or toes code No. 148-433)**

#### Treatment

- A Remove the factors which cause friction and result in the horny overgrowth

- 1 Shoes must be properly fitted
- 2 Orthopedic deformities must be treated and corrected

#### B Remove Callusities By

- 1 Paring of callus after warm water soaks
- 2 Keratolysis by use of chemical agents

a 3 Salicylic acid 4 0 3i

Acetone 4 0 3i

Collodion q s ad 1B 0 ss

Sig Apply locally to callus every night and cover with a strip of adhesive. Remove adhesive in the morning. Repeat until corn or callus is removed.

b Commercial salicylic acid corn plasters may be used

- 3 A metatarsal 1 other bar  $\frac{1}{2}$  inch wide and  $\frac{1}{4}$  inch high may be placed on the outside of the shoe just behind the weight bearing surface of the sole

### **DRY SKIN (Congenital Senile or Environmental)**

#### Treatment

- A General instruction to Patient

- 1 Avoid excessive bathing and use no soap. Avoid undue drying irritating or keratolytic medications avoid cold or dry environment
- 2 Apply simple greases liberally to the skin while it is wet
  - a Vegetable grease coconut butter vegetable cooking fats
  - b Animal greases hydrous wool fat (lanolin)
  - c Liquid petrolatum (mineral oil) and petrolatum
  - d Simple ointments (see pages 102-103)

- 3 Soap and detergents may be used when bathing but they may do more harm than good

### General Measures

- 1 Treat omphalodermatosis (e.g. a skin infection) and pyoderma by appropriate measures (see pages 72 and 85)
- 2 Vitamin A in high doses (50 000-100 000 units daily) has been recommended but results are questionable

## HERPES SIMPLEX (Cold or Fever Sore) (code No 13 166)

An acute viral infection apparently precipitated by various causes such as infection, allergy, ultraviolet radiation and psychic trauma. The small grouped vesicles appear anywhere but are most frequent on the skin and mucous membranes of the face, nose, mouth, throat and genitalia. Regional lymph nodes may be involved. Attacks are usually self-limited but are often recurrent.

### Treatment

For persistent or severe recurrent herpes

#### A General Treatment

- 1 Eliminate precipitating factors when possible
- 2 Routine smallpox vaccination at weekly intervals for 6-8 weeks. Equivocal results
- 3 Auromycin<sup>®</sup> probably effective as prophylaxis against the primary lesions

#### B Local Measures

- 1 Dust twice daily with bismuth formic acid (BF7) powder or use:
  - a. Shake lotions (see 14 15 page 100)
  - b. Spirits of camphor
  - c. Tincture of benzoin (see 47 page 105)
- 2 Hydrocortisone ointment 1-2% locally. Use as directed.
- 3 Auromycin<sup>®</sup> drops (0.5%) locally. Yes may be of value in patients with dendritic keratitis.
- 4 If there is associated cellulitis and lymphadenitis apply cool compresses
- 5 Treatment of stomatitis as outlined on page 261
- 6 Use ray therapy in selected cases. The question of immunization by expert personnel

## HERPES ZOSTER (Shingles) (code No 13 167)

An acute vesicular dermatitis of viral origin which has a characteristic distribution corresponding to the distribution of the sensory nerves and is associated with various local nerve symptoms (neuralgia, pruritus, burning and numbness) as well as motor disturbances. The involvement involves the sensory nerves of the extremities and the ophthalmic nerve most commonly (individually and unilaterally) involved but the cranial nerves may be affected (resembling chickenpox). The condition is usually self-limited and nonrecurrent, although at times a persistent or recurrent



## **III Lupus Erythematosus**

remain. The disease may be precipitated by or may be a manifestation of chronic infections, local trauma, heavy metal poisoning or lymphoblastomas.

### Treatment

#### A General Measures

- 1 Sedation. If chloralates or bromids may help control tension and nervousness associated with neuralgia.
- 2 Analgesics. Aspirin 0.63 Gm (10 gr) or aspirin compound with or without codeine phosphate 0.63 Gm (1 gr) will usually control the pain.

#### B Local Measures

- 1 Wet dressings may be necessary for acute and extensive inflammatory lesions (see pages 93-99).
- 2 Calamine lotion or other astringent lotions (see page 100) are often of value. Apply lotion liberally and cover with a protective layer of cotton batting. Avoid greases.
- 3 X-ray therapy given by an expert may be helpful.
- 4 ACTH gel, 40 mg i.m. daily for 3 days may relieve the pain.

## **LUPUS ERYTHEMATOSUS (code No. 110 1x9)**

### Diagnosis

An acute or chronic dermatitis of unknown origin manifested by two main clinical types:

- A Discoid Type. Mild local, chronic eruption over nose and cheeks (butterfly pattern) with no constitutional symptoms.
- B Disseminated Type. A serious systemic disease which occurs in acute and chronic forms with or without discoid skin lesions and associated with fever, weakness, anemia, and evidence of diffuse vascular lesions such as endocarditis, arthritis, and nephritis (see page 319 for diagnosis and treatment of disseminated type).

### Treatment

#### A General Measures

- 1 Perform complete medical study to rule out systemic lupus erythematosus.
  - a Examine for chronic infections.
  - b Determine cardiac, renal, and joint status.
- 2 Provide protection from sunlight and all other powerful radiation. Do not use any form of radiation therapy.
- 3 Maintain optimal general health by well-balanced adequate diet with supplementary vitamins and iron as indicated. Insure adequate rest and prescribe bed rest when the patient is febrile.
- 4 Nonspecific therapy for discoid type only.
  - a Quinacrine hydrochloride (Atabrine®) 0.3 Gm (5 gr) orally daily for 2 weeks then 0.1 Gm (1½ gr) daily for 3 months or more.
  - b Chloroquine diphosphate 0.5 Gm (7½ gr) daily for 1 week then 0.3 Gm (3¾ gr) daily watch for signs of toxicity with both of these drugs.

- B Local Measures. Treat the existing stage of dermatitis by appropriate measures (see pages 96-97 and 108).

## INFECTIONS OF THE SKIN

### ACUTE SUPERFICIAL INFECTIONS

1 Impetigo contagiosa (code No 111 10 )  
2 Ecthyma (code No 110 105 )  
3 Syphilis (code No 110 105 )  
4 Acute superficial infections

- LOCAL INFECTIONS**
- acute superficial infections include the following
- 1 Impetigo contagiosa (code No 111 10 )
  - 2 Ecthyma (code No 110 103 1)
  - 3 Erysipelatous (code No 161 105)
  - 4 Acute infectious eczematoid dermatitis (code No 110 1003)
  - 5 Simple superficial pyoderma (code No 1 100 1)
  - 6 Secondary infection of other dermatoses
- The offending organism is usually hemolytic Staphylococcus aureus and/or the streptococcus
- Treatment
- A G

T ment

Systemic anti-infectives may be tried if the skin infection is resistant to local treatment if this is accompanied by fever, if it is complicated or if it involves the so-called danger areas of upper lip, nose and eyes. (Refer to section on Antibiotic Therapy on page 514)

Penicillin in a daily dose of 300,000 units (in 4 doses) is effective for this purpose but may be modified in the case of other antibiotic drugs may be substituted as follows:

Local Measures

1. Cleanse gently with water.
2. Soaks or compresses.

1. Cleanse gently with mild solution of soap and water (see pages 98-99) to involve the skin is softened by soaking and trim away necrotic tissue. Local anti-infective agents are of benefit individually until all wound is healed.

- 1 Cleanse gently with mild solution of cast and water  
2 Soaks or compresses to involve d a 15 min tes b i d  
(see pages 98 99)  
3 When skin is softened by soak  
4 Local anti infecti e agents are of p d tu These may  
be i i d individually until effe tiv ag nt i d terminated  
all wing 3 4 d ys for evaluation They should be ppli d  
initi ly at night and protected by dr esing socks should be  
ppli d during the day After the ea ha l d any i  
th e pr p rations may be ppli d 2 4 tim s d ly  
a Aqueous omycin 1% locally q i d  
b Vi f m e oint nt (iodochlorhyd oxyquin lin ) 3%  
locally b i d  
Other nitri ls s m e or in comb  
locally b i d to e i f  
A eom

Other antibiotics are also used in combination as outlined locally b i d to q i d Th included T ramycin® A. someycin® and polymyxin B in combination with bacit cin or T mycin® neomycin hlor mph ni c and erythromy in (s page 514) al agents ar of alve in s d i k of

- [illegible]

## III Skin Infections

### b Sulfathiazole Urea powder

- (1) R Sulfathiazole powder TO O 211 1/2  
Urea 30 O 31

Sig Dust or rub in a small quantity locally

- (2) Sulfathiazole 5% in starch lotion (R 15 page 100)

Shak well and apply locally to involved area

### Prophylaxis

Control precipitating or aggravating factors Systemic causes (e.g. diabetes) or local causes (e.g. mechanical or chemical skin irritations, discharges, etc.)

## CHRONIC SECONDARY INFECTIONS

Determine all possible factors favoring chronicity Obtain bacterial cultures and determine organism sensitivity to antibiotic agents whenever possible

### Treatment

#### A General Measures

- 1 Diet Well balanced and adequate in proteins and vitamins
- 2 Consider use of vigorous systemic anti-infective therapy

#### B Local Measures

- 1 Use local measures as for cut superficial infections
- 2 Treat underlying dermatosis according to stage and type of lesion (see pages 86-97 and 103)
- 3 Consider x-ray therapy if all other measures are ineffective  
This must be reserved for the specialist

## ACUTE and CHRONIC INFECTIONS of SKIN APPENDAGES

Examine for local and systemic causes of these infections particularly if they become severe or chronic The following disorders are included

- 1 Folliculitis pustular (code No 181.9x2)
- 2 Furunculosis (code No 181.100.0)
- 3 Carbuncle (code No 181.100.3)
- 4 Hidradenitis (code No 182.100)

### Treatment

- A General Measures Use systemic anti-infective therapy if lesions are severe, extensive, complicated or located in dangerous areas (throat, neck and head)

#### B Local Measures

- 1 Avoid over-manipulation of inflamed areas
- 2 Use moist or dry heat to help larger lesions localize
- 3 Use proper surgical incision, epilation or debridement after lesions are mature

## COMPLICATIONS OF SKIN INFECTIONS

If pathogenic bacteria from infections of the skin invade deep structures a number of the following may be produced and other more serious infection may also occur

- 1 Cellulitis (code No 18 100)
- 2 Acute lymphangitis (code N 54 100 1)
- 3 Acute lymphadenitis (code No 55 100 1)

### Treatment

#### A General Measures

- 1 Bed rest with immobilisation of affected extremity or part
- 2 Systemic anti-infective agent must be administered in effective doses (see page 514)
- 3 Analgesic necessary for pain (see page 36)

#### B Local Measures

- 1 Immobilisation of affected part in lightly elevated position
- 2 Local hygiene using warm moist compresses if abscesses or pustules are present. Avoid maceration of skin (use no occlusive covering)
- 3 Local anti-infective agents to open infected areas at night

## FUNGAL INFECTIONS OF THE SKIN

### GENERAL CONSIDERATIONS

#### Diagnosis

Usually based on

#### A Characteristic clinical features of lesion (See description below)

#### B Laboratory Examination

- 1 Direct examination of fungi in 10% potassium hydroxide preparation (acquires from suspected lesions)
- 2 Culture of organisms
- 3 Skin tests (e.g. Trichophyton) are not reliable
- 4 Staining of histological sections with periodic-acid Schiff technique

#### Treatment

#### A Local Measures

- 1 Treat cutaneous fungal infections initially as if primary cutaneous dermatitis (see page 108) it may be necessary to treat the dermatitis before instituting fungicidal medication
- 2 Most fungi are local and long skin irritants. It is easy to overtreat. AVOID this

#### B General Measures and Prophylaxis

- 1 Keep kindly moist skin favour the growth of fungi
- a Cool limit when possible is to be preferred
- b Emollient preparation must be avoided. Redness and irritations in hot of day
- c Dry thoroughly after bathing or after perspiring
- d Socks and other clothing should be changed often

## 22 Tinea Capitis

- e Sandals or open toed shoes should be worn as they permit adequate drying of feet
- f Secretions of skin should be reduced or controlled
  - (1) General or systemic measures
    - (a) Sedatives in tense nervous patients Phenobarbital, 15-30 mg ( $\frac{1}{4}$  to  $\frac{1}{2}$  gr) t i d to q i d
    - (b) Anhidrotic drugs (e.g. atropine) are usually ineffective
  - (2) Local measures
    - (a) Talk or other drying powders (see page 89)
    - (b) Drying soaks (see pages 88-89)
- g Toughen skin by graded daily sunbaths or by quartz lamp treatment
- 2 Feet of fungal infections should be eradicated or controlled
  - a Treat nails umbilicus groin webs of toes and other areas where fungi are found
  - b Group or community showers or bathing places unless strictly supervised should be avoided

### TINEA CAPITIS (Ringworm of Scalp) (code No 162 211)

This contagious sometimes epidemic condition occurs almost exclusively in children. It is very persistent but clears spontaneously at puberty. The lesions are originally red and scaling and result in circular areas of alopecia. Fluorescence under the Wood light is characteristic in Microsporon infections (90% of cases). There is often a history of contact with infected individuals or household pets.

#### Treatment

- A General Measures None
- B Local Specific Measures
  - a It may require 2 months or more to cure the disease. The human type is more difficult to cure than the animal type (dogs and cats).
  - 1 Scalp cleansing and preparation (Not essential.)
    - a Clip hair closely every 2 weeks and have patient wear a clean stocking cap or skull cap for protection
    - b Wash scalp as necessary
  - 2 Fungicidal salves. Rub these well into scalp morning and night after scalp has been washed.
    - a Salicylanilide 3% in Carbowax 1500<sup>®</sup> ointment
    - b Benzoic acid and salicylic acid ointment (Whitfield's) one-half strength (R 34 page 104)
    - c Sulfur salicylic acid ointment (R 38 page 104)
  - 3 Epilation. Use Westinghouse 250 Watt purple X lamp to demonstrate fluorescent infected hairs. Remove infected hairs daily by tweezer or by adhesive tape technique.
  - 4 X-rays may be used effectively and may work when chemical and mechanical measures fail. X-ray therapy must be given by trained personnel only. Do not re-epilate with x-rays.

Trophylia is

A Indicated

- 1 Exchange of bedding must be avoided

- 2 Infected individuals or household pet must be vigorously treated and re-examined for determination of cure
- 3 Scalp must be washed after barber shop haircut

**B Group**

- 1 Routine school surveys may be advisable
- 2 Epidemic precautions
  - a Wood light examination of students less than 13 years old
  - b Isolation of infected individual in special classrooms
  - c Careful follow-up of infected individuals and periodic re-examination of all children until all cases are cured
  - d Education of barbers regarding handling of infected individuals

**PITYRIASIS VERSICOLOR OR TINEA VERSICOLOR**  
(code No 112 208)

A mild condition characterized by tan or pinkish, very fine scaling on areas of variable size only mildly pruritic usually found on the upper trunk. It also occurs on the face and is depigmented for a few months.

**Treatment**

- A General Measures Encourage normal skin hygiene
- B Specific Measures One of the following may be used
  - 1 Sodasol 1% in 10% aqueous solution bid
  - 2 Mild White lead ointment 1/4 to 1/2 strength (B 34 p 104) bedtime

**TINEA CORPORIS OR TINEA CIRCINATA**  
(Body Ringworm) (code No 130 311)

Body ringworm is characterized by single or multiple (relative to few) slightly pale circular lesions with clear central area and with minute vesicles. In the actively expanding periphery they are found most commonly on the trunk, neck and limbs. Lesions occasionally show thick pigmentation. Diagnosis should be confirmed by demonstration of the fungi.

**Treatment**

- A General Measures (See page 87)
- B Local Measures Avoid oral treatment
  - 1 Treat the proper stage of the material (see 98 to 99 108)
  - 2 Fungicidal agents
    - a 5% Icthyol ointment 0.3 gr w
    - 5% Mercurochrome 0.5 gr x
    - Hydrophilic ointment
    - q.s. ad 30 g i
    - 3% Locally bid
  - b Zincundecate (undecylic acid and zinc undecylate) in the less chronic and non-inflamed lesions

**Prevention**

- A General Measures on page 87
- B Avoid contact with infected household pet
- C Avoid hanging of clothing without detergent laundry ring

# USEFUL MEDICATIONS FOR SKIN DISEASES

THE MEDICAL OFFICE OF THE ARMY

Name	Action	Preparation	Technique
A Sarc and soda bath	Cleansing and soothing	1/2 cup hydrous sulfide and 1/2 cup soda	No anesthetic solution is used. It is used for relief of itching.
B Cool wet dressings	Cooling and soothing	1 To 10 parts of water and 1 part of alcohol	Use in cases of acute inflammation and itching. It is used for relief of itching.
b Alum Acetate U.S.	Cleansing and soothing	1/2 cup hydrous sulfide and 1/2 cup soda	No anesthetic solution is used. It is used for relief of itching.
c Potassium Permanganate U.S.	Disinfectant	1/2 cup hydrous sulfide and 1/2 cup soda	No anesthetic solution is used. It is used for relief of itching.
C Hot wet dressings	Relieves itching and inflammation	1 To 10 parts of water and 1 part of alcohol	Use in cases of chronic inflammation and itching. It is used for relief of itching.
D Starch U.S.P. (Add 5% solution of coal tar)	Soothing and drying	1/2 cup hydrous sulfide and 1/2 cup soda	No anesthetic solution is used. It is used for relief of itching.

N m	A t	P s p m o	T h n c
E Hyd ophal Ointm t U S P	V h l f w t l b l m d F p b h F g d F m	O m y d d 5% m m t d m y 1% a l y l d 3% u l f u 5% d t r g n t l t i f l t	Apply sp i gly w th fingertip b i d
F 5% S : ylanil d N F in C bowak 1500 <sup>3</sup>	F o n p t e	D p n 60 Gm o b ( u n t m t )	L ally b d to t l i p (l t a o t e r y t o c l p t h h i )
G Kw l p <sup>3</sup> Intm t (h hlo y l h a n e)	F r e b l e d p d u l i s	D p 30 Gm	L ally b d f o l t 3 d y s
H Aq m y in (0 l e)	F p y o d m	R N m y n 0 12 g r i D t l l e d w t q d 120 0 i	Apply with c t t b d to q i d
I Methyl l n Chlo d U S P (G n t l a n i l t s)	F o m m l i	D p e 30 i o f 1 7 a q o s l t	P l n t o with p p l c a t o r o e d l y
J P a s m b o c A d U S P e t m u l o m b (10 <sup>4</sup> )	P t t f m t i o n r y s	D e p n e 50 Gm o	Apply t m p o d u f a s a c h m o r n i n g
K L a P t (Z O d P t N P)	F t t a n d a t h i n g	D p s 30 Gm i	Locally b i d



# METHODS OF LOCAL TREATMENT OF VARIOUS TYPES OF SKIN LESIONS

Type of Skin Lesion	Example	Methods of Local Treatment Always use the same as well as type of dermatitis
1. Maculæ Simple erythema (asymptomatic) Pruriginous erythema	Drug erythema Sunburn	Soothing wet dressings or stake lotions
2. Maculopapular lesions Papulosquamous Lesions Acute Chronic Acanthosis Lichenified lesions Verrucous lesions	Pityriasis rosea Psoriasis Psoriasis Acne vulgaris Lichen planus Verru vulgaris	Mild keratoplasty lotions and ointments Soothing wet dressings or stake lotions Keratoplastic and later keratolytic agents Herbolytic and astringent agents Keratoplasty and later keratolytic agents Keratolytic and caustic agents
3. Multiple vesicles or diffuse weeping Lesions Erythematous lesions Ulcers	Eczema Itchy scaly Pruriginous	Soothing wet dressings during daytime and stake lotions or pastes at night. Asper as a substitute change to lotions and creams Shake lotions or wet dressings Wet dressings and bed linens have lotions
4. Impetigo Ecthyma Furunculosis Pyoderma	Impetigo Ecthyma Furunculosis Pyoderma	Wet dressing and bed linens antiseptic powders and ointments Wet dressing and bed linens antiseptic powders and ointments Wet dressing and bed linens antiseptic powders and ointments Wet dressing and bed linens antiseptic powders and ointments

Type of Skin Lesion	Example	Always treat it as well as type of dermatitis
5 Ulcers Simple superficial Deep pyogenic Diptheritic Simple whitish Anginofuritic Dermatitic	Simple Ointment Topical ulcer Topical	Wet dressing anti-infective solutions and ointments Wet dressing anti-infective solution and ointments Wet dressings anti-infective solution and ointments Antipruritic cooling baths and skin lotions
6 Ulcers Simple whitish Anginofuritic Dermatitic	HIV Anginofuritic Simple furitic	Antipruritic cooling baths and ointments (acid wet Sulfuric acid solution) Wet dressing debridement follow with skin lotions and greases Wet dressing debridement follow with anti-infective solution and ointment
7 Eczema Eczematoid Eczematoid Infected	Eczema Impetigo	Keratolytic and interkeratolytic agents Wet dressing bath skin lotions and later greases Wet dressing less ointments Keratolytic agents acid plastic and products
8 Dermatitis Adherent Non-adherent Chronic Acute	Psoriasis Erythematous Dermatitis Scabious Intertrigo	Wet dressing acid plastic and products Wet dressing acid plastic and products Wet dressing acid plastic and products Wet dressing acid plastic and products
10 Malignant		

## SIMPLE SOLUTIONS FOR SOAKS AND WET DRESSINGS

Indications for use of each solution are given in the following table.

(1) Solutions must be applied cool (but for infections).

(2) Wet dressings (2-3 quarts of solution) for heat and for 1-2 hours.

(a) Open dressings for local dilation, wet towels, heat, and with solution.

(b) Open dressings for vascularity and when marked and each of the above.

For quantities are necessary for the above.

(c) Coated dressings should not be used.

The following table gives the composition of the solutions.

Agent	Action	Preparation	Concentration	Preparation
(See above)	(See above)	(See above)	(See above)	(See above)
1. Sodium Chloride U.S.P.	(See above)	0.1000 (0.1%)	0.9%	2-4 dr (2 tsp) NaCl to 1 qt water or 30 Gm NaCl to 1 liter water
2. Sodium Bicarbonate U.S.P.	Antipruritic	1.50 (1.5%)	3.0%	2-4 dr (2 tsp) NaHCO <sub>3</sub> to 1 qt water or 30 Gm NaHCO <sub>3</sub> to 1 liter water
3. Boric Acid U.S.P.	Antipruritic	1.0 (1.0%)	3.0%	2-4 dr (2 tsp) H <sub>3</sub> BO <sub>3</sub> to 1 qt water or 30 Gm H <sub>3</sub> BO <sub>3</sub> to 1 liter water
4. Magnesium Sulfate U.S.P.	Antipruritic	1.0 (1.0%)	3.0%	2-4 dr (2 tsp) MgSO <sub>4</sub> to 1 qt water or 30 Gm MgSO <sub>4</sub> to 1 liter water
5. Aluminum Subacetate U.S.P.	Antipruritic	1.200 (1.2%)	5.0%	4 Dm borate to 1 qt or 50 = 1 liter water
6. Silver Nitrate U.S.P.	Antipruritic	1.10 (0.11%)	1.400 (0.14%)	10 cc of 25% AgNO <sub>3</sub> solution or 5 Gm AgNO <sub>3</sub> to 1 qt = 1 liter water

Agent	Active Ingredient	Range of Concentration (%)	Molecular Weight	Preparation of Solution
R 1 Mercury Biphosphate	Antiseptic	1:10,000 to 1:1,000 (0.01% to 0.1%)	33,000	On 10 Gm (15 gr) tablet HgCl <sub>2</sub> to 1 qt of water or of 50% alcohol. Do not use on denuded areas.
R 2 Potassium Permanganate	Antiprotozoal, Oxidizing, Antiseptic	1:10,000 to 1:400 (0.01% to 0.25%)	158,000	On 0.3 Gm (5 gr) tablet KMnO <sub>4</sub> to 1 qt of 3% formalin or 0.1 Gm (1 1/2 gr) KMnO <sub>4</sub> to 1 qt of liter water.
POWDERS				
Agent	Active Ingredient	Range of Concentration (%)	Molecular Weight	Preparation of Solution
R 3 Silver Nitrate	Antiseptic	1:10,000 to 1:1,000 (0.01% to 0.1%)	33,000	On 10 Gm (15 gr) tablet HgCl <sub>2</sub> to 1 qt of water or of 50% alcohol. Do not use on denuded areas.
R 4 Talcum	Antipruritic	1:10,000 to 1:400 (0.01% to 0.25%)	158,000	On 0.3 Gm (5 gr) tablet KMnO <sub>4</sub> to 1 qt of 3% formalin or 0.1 Gm (1 1/2 gr) KMnO <sub>4</sub> to 1 qt of liter water.
R 5 Zinc Oxide	Antipruritic	1:10,000 to 1:400 (0.01% to 0.25%)	158,000	On 0.3 Gm (5 gr) tablet KMnO <sub>4</sub> to 1 qt of 3% formalin or 0.1 Gm (1 1/2 gr) KMnO <sub>4</sub> to 1 qt of liter water.
R 6 Zinc Oxide	Antipruritic	1:10,000 to 1:400 (0.01% to 0.25%)	158,000	On 0.3 Gm (5 gr) tablet KMnO <sub>4</sub> to 1 qt of 3% formalin or 0.1 Gm (1 1/2 gr) KMnO <sub>4</sub> to 1 qt of liter water.
R 7 Zinc Oxide	Antipruritic	1:10,000 to 1:400 (0.01% to 0.25%)	158,000	On 0.3 Gm (5 gr) tablet KMnO <sub>4</sub> to 1 qt of 3% formalin or 0.1 Gm (1 1/2 gr) KMnO <sub>4</sub> to 1 qt of liter water.
R 8 Zinc Oxide	Antipruritic	1:10,000 to 1:400 (0.01% to 0.25%)	158,000	On 0.3 Gm (5 gr) tablet KMnO <sub>4</sub> to 1 qt of 3% formalin or 0.1 Gm (1 1/2 gr) KMnO <sub>4</sub> to 1 qt of liter water.
R 9 Zinc Oxide	Antipruritic	1:10,000 to 1:400 (0.01% to 0.25%)	158,000	On 0.3 Gm (5 gr) tablet KMnO <sub>4</sub> to 1 qt of 3% formalin or 0.1 Gm (1 1/2 gr) KMnO <sub>4</sub> to 1 qt of liter water.
R 10 Zinc Oxide	Antipruritic	1:10,000 to 1:400 (0.01% to 0.25%)	158,000	On 0.3 Gm (5 gr) tablet KMnO <sub>4</sub> to 1 qt of 3% formalin or 0.1 Gm (1 1/2 gr) KMnO <sub>4</sub> to 1 qt of liter water.
R 11 Zinc Oxide	Antipruritic	1:10,000 to 1:400 (0.01% to 0.25%)	158,000	On 0.3 Gm (5 gr) tablet KMnO <sub>4</sub> to 1 qt of 3% formalin or 0.1 Gm (1 1/2 gr) KMnO <sub>4</sub> to 1 qt of liter water.
R 12 Zinc Oxide	Antipruritic	1:10,000 to 1:400 (0.01% to 0.25%)	158,000	On 0.3 Gm (5 gr) tablet KMnO <sub>4</sub> to 1 qt of 3% formalin or 0.1 Gm (1 1/2 gr) KMnO <sub>4</sub> to 1 qt of liter water.
R 13 Zinc Oxide	Antipruritic	1:10,000 to 1:400 (0.01% to 0.25%)	158,000	On 0.3 Gm (5 gr) tablet KMnO <sub>4</sub> to 1 qt of 3% formalin or 0.1 Gm (1 1/2 gr) KMnO <sub>4</sub> to 1 qt of liter water.

**DISPATCHING**

2000

- 1 To correct fat deficit wcy in a dry skin
- 2 To provide mechanical protection to the underlying lesions
- 3 To help ab orb or limb be transudates from underlying lesions (This holds true only for the hydrophilic preparation )
- 4 To apply a thin discinal at risk to th skin

† A wet influence of winged hairs  
Hairy areas (e) of the pygidial  
fe parietal

Preparation	Preparation	Preparation
<b>ointments</b>		
R 4 Petrolatum white U S		
R 2 White soft paraffin B I		
R 2 Petrolatum white U S		
R 26 Wool fat hydrous U S		
R 27 Wool fat U S		
R 28 Zinc oxide U S		
R 29 Th eobrom oil U S		



## OINTMENTS, MISCELLANEOUS STANDARD PRESCRIPTIONS

Common Name	Preparations	Instructions and Remarks
R 34 Benzocaine Salicylic Acid V P (Whiffle)	Salicylic acid Benzocaine Wool fat Petrolatum q s ad	Sig Apply locally to skin p r n Remarks Effective for fungicidal combination preservative by 14 days
R 35 Aluminum acetate ointment (1233)	Alum acetate Sol Wool fat Zinc oxide paste	Sig Apply locally to skin p r n Remarks Valuable for reducing inflammation
R 36 Sulfur salicylic acid ointment	Sulfur Salicylic acid Petrolatum q s ad	Sig Apply locally to skin p r n Remarks Excellent fungicidal combination
R 37 Calamine cream	Hydrophilic ointment U & P Calamine lotion	Sig Apply locally to skin p r n Remarks Good general purpose cream Useful vehicle for water soluble germs
R 38 Ammonium mercury ointment	Ammonium mercury Petrolatum q s ad	Sig Apply locally to skin p r n Remarks For borthele dermatitis and psoriasis
R 39 Kaolin and sulfur ointment	Kaolin Sulfur ppt Zinc oxide oint q s ad	Sig Apply locally to skin Remarks A good substitute exfoliating paste for psoriasis
R 40 Hexachlorocyclohexane (Kw 115) ointment	Kw 115 ointment	Sig Apply as directed Remarks A good substitute for hexachlorocyclohexane

## SOLUTIONS TINCTURES AND PAINTS

Name	Preparation	Remarks
R 41 Methylene Blue (Glycerin Sol)	10% aqueous solution	Anti-pyretic (gram positive germicide) and fungicide (fungal)
R 42 Sodium Chloride USP	10% aqueous solution	Fungicide (epiphytic)
R 43 Silver Nitrate USP	10% aqueous solution	Useful for treating and preventing
R 44 Chlorine USP	4% in hydrochloric acid	Fomoricidal preparation
R 45 Nitric Acid (Mist)	0.5% (1:200) solution	Useful for treating and preventing
R 46 Alcohol (Mist)	85% alcohol, 15% acid	Apply to allylic lesions
R 47 Benzoic Acid Compound Tincture USP	10% alcohol, 10% acid	Effective fungicide (combination)
R 48 Soft Soap Liniment USP	10% alcohol, 10% acid	May be used by immersion in alcohol
R 49 Tincture of Iodine	10% alcohol, 10% acid	Useful for treating lesions
R 50 Cetyl Alcohol USP	10% alcohol, 10% acid	Useful for treating lesions
R 51 Soft Soap Liniment USP	10% alcohol, 10% acid	Add up to 1 part of water to make emulsion
R 52 Tincture of Iodine	10% alcohol, 10% acid	Useful for treating lesions
R 53 Cetyl Alcohol USP	10% alcohol, 10% acid	Useful for treating lesions
R 54 Soft Soap Liniment USP	10% alcohol, 10% acid	Add up to 1 part of water to make emulsion
R 55 Tincture of Iodine	10% alcohol, 10% acid	Useful for treating lesions
R 56 Cetyl Alcohol USP	10% alcohol, 10% acid	Useful for treating lesions
R 57 Soft Soap Liniment USP	10% alcohol, 10% acid	Add up to 1 part of water to make emulsion
R 58 Tincture of Iodine	10% alcohol, 10% acid	Useful for treating lesions
R 59 Cetyl Alcohol USP	10% alcohol, 10% acid	Useful for treating lesions
R 60 Soft Soap Liniment USP	10% alcohol, 10% acid	Add up to 1 part of water to make emulsion
R 61 Tincture of Iodine	10% alcohol, 10% acid	Useful for treating lesions
R 62 Cetyl Alcohol USP	10% alcohol, 10% acid	Useful for treating lesions
R 63 Soft Soap Liniment USP	10% alcohol, 10% acid	Add up to 1 part of water to make emulsion
R 64 Tincture of Iodine	10% alcohol, 10% acid	Useful for treating lesions
R 65 Cetyl Alcohol USP	10% alcohol, 10% acid	Useful for treating lesions



DERMATOLOGIC MEDICAMENTS

The following are the most commonly used in the treatment of the skin. In the following list are given 100 and 102. In general, the preparations are simple and are easily absorbed. The following are the most commonly used in the treatment of the skin. In the following list are given 100 and 102. In general, the preparations are simple and are easily absorbed. The following are the most commonly used in the treatment of the skin. In the following list are given 100 and 102. In general, the preparations are simple and are easily absorbed.

Preparation	Indication	Concentration	Form	Notes
(For eczema and other skin diseases) The use of art by drying and drying the skin				
Jo	eczema	10%	ointment	
102	eczema	10%	ointment	
100	eczema	10%	ointment	
101	eczema	10%	ointment	
103	eczema	10%	ointment	
104	eczema	10%	ointment	
105	eczema	10%	ointment	
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199	eczema	10%	ointment	
200	eczema	10%	ointment	



# GENERAL RULES GOVERNING CHOICE OF TREATMENT OF VARIOUS STAGES OF DERMATOSES

Recommended Medicaments		Page
Wet preparations	Soaks For lesions localized to extremities	92
	Wet dressings For localized lesions of head, neck, trunk or extremities	93
	Baths For generalized lesions	97
	Subacute lesions	109
Chronic lesions	Emulsion	100
	Hydrophilic ointment	103
	(Highly medicated cream)	103
	Creams (not in water) cream	103
Greasy ointment		104

## ACUTE LESIONS

Characteristics:  
Recent onset and  
burning, swollen  
itching, blistering,  
and oozing

## SUBACUTE LESIONS

Characteristics:  
Intermediate duration  
subsiding lesions and  
less angry in appearance

## CHRONIC LESIONS

Characteristics:  
Longer duration  
quiescent thickening,  
cracked fissured  
and scaly

Start directions for choice of treatment will vary with the individual case. Also will depend upon a wide variety of factors including a consideration of the dermatosis, extent of lesions, general character of patient's skin, previous medication and drug allergies.







and accessory structures. These usually require antibiotics or sulfonamides and may require the attention of an otolaryngologist.

### Etiology

The principal predisposing factor is similar to that of any contagious disease: avoidance of exposure whenever possible; avoidance of sudden changes of temperature and excessive fatigue. Administration of large doses of any of the vitamins, cold vaccines orally or by injection (gamma globulin or "hardening up") have all proved valueless in preventing or in altering the course of the disease.

## ACUTE SINUSITIS (code No. 32-130)

The acute infection of the paranasal sinuses following upper respiratory infections is usually caused by a secondary bacterial invader. The invading organisms most frequently are streptococci, staphylococci, or pneumococci.

### Treatment

#### A. Specific Measures

1. Penicillin is the drug of choice since most of the organisms are penicillin sensitive. It is administered as follows: 300,000 units penicillin procaine i.m. once or twice daily. Other wide spectrum antibiotics may also be employed.
2. Local administration of penicillin and other antibiotics by nose drops and use of negative pressure is still difficult to evaluate.

#### B. General Measures

1. Bed rest.
2. Local external heat over the sinuses.
3. Analgesics. Aspirin or codeine may be used.
4. Vasoconstrictor drugs. Non-irritating nose drops may be used to facilitate drainage or drugs may be given in tablet form by mouth for similar effect (see page 110).

#### C. Do not use steam inhalation in sinuses during acute sinusitis

## EPISTAXIS (code No. 301)

Epistaxis or nosebleed may be due to a variety of diseases or disorders.

- #### A. Predisposing Factors
- Blood dyscrasias, hypertension, arteriosclerosis, prothrombin deficiency (e.g., cirrhosis of the liver), nasal ulceration, nasal angioma, and certain of the infectious diseases (e.g., measles and rheumatic fever).

- #### B. Precipitating Factors
- External trauma to the nose, violent blowing of the nose, sneezing, picking of nose, increase of existing high blood pressure or lowering of atmospheric pressure.

- #### C. Location
- The bleeding site is most frequently on the anterior portion of the nasal septum, less often at the end of the inferior and middle turbinates, and rarely further posteriorly.

Tr tra nt

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■ oxid w ll usu lly top the bleeding
- 2 ■ te ation Wh n active bl eding h s e sel to chi g  
the bl eding point with be d of hromium tri xid (chromic  
d) t hlo cet a id w ll u lly pre nt furth r  
ble ding El ctro at y i ls ti f ct y
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b ont all d w th a tampon int odu d th ough th tril  
If bl eding i po t ri it may b se ssa y to intr du a  
post or nasal pack This i done by the use of two st ing  
att hing on string ne ■ nd of a oiled 2 x 2 o 4 x 4  
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p d A soft athet ri then introduced into the n soph ynx  
through ■ no tr l and pull d out through the r uth Th  
e d of one f the f st two st ings i tied to th o l po tion  
of th catheter and pulled b ck through the st l Th  
tring a th rem v d from the cath ter and the procedure  
r p ated p lling th ond string through th oth po tril  
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th tied v a p d under the nasal e ptum Th th rd  
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Do tle g k m m r than 48 hour
- 4 l p k on th no ort the b k f ne k are f no b efit
- B Sp fi M ■ Tre t the und ly g d l as

**ACUTE TONSILLITIS (code No 834 100)**

Acute t nsillit is an inf cti of th f lal tonsils ca s d by  
any of a number of o gan ms It i b ra t i d by both loc l and  
g n raliz d sympt m f varying d gr

T tm nt

D p ds on the usual gni m (Fo at pt o so  
th o t a pag 459)

**ALLERGIC RHINITIS (Hay Fever) (code No 310 392)**

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ey Pollens a th m t common all g ms

Tr tm nt

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s ptibl ind vidu l Fo b t sult the psych ld be  
started 3 6 month b f e th ons t of the hay fev r ason o





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(s page 110)

d S d t om may be of v l e f p t nt i ne you o up t  
(se pag 35)

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## DISEASES OF THE BRONCHI

### ACUTE TRACHEOBRONCHITIS

(Tracheitis code No 340 100) (Bronchitis code No 350 100)

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- 3 Sleeplessness Pentobarbital Sodium ■ S P Pentobarbitone Sodium ■ P 0 1 Gm (1½ gr) at bedtime should be given

### CHRONIC TRACHEOBRONCHITIS (Chronic Bronchitis code No 350 100 0)

A chronic nonspecific inflammation of the tracheobronchial tree manifested by cough which is usually the only constant symptom. The cough may be productive or non-productive. *Do not diagnose chronic bronchitis on the basis of chronic cough alone. Any patient with a chronic cough should be given a thorough examination including a chest x-ray.* As a rule a diagnosis of chronic bronchitis can be made only by exclusion. Primary chronic bronchitis is a rare disease almost all cases of chronic bronchitis are secondary to other respiratory conditions or inflammations. Intractable cases may have an allergic basis. Physical findings may be absent or a few rhonchi and wheezes may be heard.

#### Treatment

A Specific Measures There is no specific treatment for chronic bronchitis treat the underlying condition

#### B General Measures

- 1 Remove or eliminate exciting causes such as smoking excessive cold air damp atmosphere industrial fumes etc
- 2 Drugs
  - a Ephedrine and similar drugs may give relief in many cases when bronchospasm is present
  - b Saturated solution of potassium iodide 5-10 drops t.i.d. as tolerated may be helpful
- 3 Adequate rest in a dust free room
- 4 Optimum nutrition and hygiene

### BRONCHIAL ASTHMA (code No 350 390)

Bronchial asthma is a symptom complex due to a variety of causes. It is characterized by dyspnea, especially of the expiratory phase with wheezing and whistling which are due to edema of the bronchial lining and/or contraction of the smooth muscles leading to constriction of the bronchi.

#### Diagnosis

A History of repeated attacks of expiratory dyspnea frequently in an emotionally labile patient with a definite allergic tendency. The attacks are precipitated by exposure to the allergen or at times by severe emotional stress.

#### B Physical Examination

- 1 During typical attack Examination is characteristic
  - a Severe expiratory dyspnea and at times cyanosis
  - b Chest held in partial inspiratory position
  - c Inspiration short and expiration greatly prolonged
  - d Cough difficult and may become violent
  - e Sputum thick and tenacious
  - f Chest hyperresonant to percussion

g Ch at I II ■ musical rules which frequently may be hard at a distance

2 ■ between attacks May be entirely negative or may show a very full emphysematous chest with reduced vital capacity

C Diff ti I D g col Many diseases may simulate bronchial asthma Of these the most important are cardiac asthma and renalized emphysema specially where chronic bronchitis is superimposed The appearance of bronchial asthma in mild disease should make one suspect bronchial asthma

## Treatment

A The treatment may be divided into two phases

1 Treatment of the acute attack

2 Inter-attack prophylaxis aimed at preventing further attacks

B Drugs used for the specific treatment of asthma In the past little faith was put in the possibility that drugs have come to be looked upon as specific for relief while other preparations of value in assisting the attack It is necessary to know the properties available their modes of administration and the indications for their use The chapter page 118 summarizes the important drugs

Epinephrine (adrenaline) is the drug of choice for the emergency management of acute bronchial asthma However it has been shown that it is of opinion (ACTH) or cortisone can be used to stop an attack when the epinephrine measure fails The onset of action of ACTH is so slow is much lower than that of epinephrine but they should be employed concurrently with epinephrine in severe attacks of asthma Epinephrine must be used cautiously in patients with cardiac asthma hypertension or angina

## Treatment of the Acute Attack Do not use morphine

A Mild Moderate Attack Epinephrine (adrenaline) is the drug of choice

1 Epinephrine injection (injection of adrenaline) 0.2 to 0.5 cc (3 to 7 min) of 1:1000 subcut

2 Epinephrine inhalation (1 to 2 mg aqueous solution) by nebulizer very 30 to 60 minutes per day

3 Moderate Attack Repeat epinephrine (adrenaline) subcut every 1 to 2 hours

4 Epinephrine inhalation 0.2 to 0.5 (3 to 5 min) 1:1000 1 M may also be given at onset if a prolonged effect is desired May repeat in 10 to 15 minutes

5 Aminophylline (theophylline ethylenediamine) 0.24 to 0.48 Gm (3 3/4 to 7 1/2 g) in 10 to 20 cc (2 1/2 to 5 dr) as 1 to 2 wly I V If it is not available 0.48 Gm (7 1/2 g) may be added to 500 to 1000 cc of saline and given by I V drip May also give the same result by inhalation or orally prior to giving the same dose

6 Ephedrine Hydrochloride 25 to 50 mg (3/8 to 3/4 gr) with water tabs at my elbow would be (see page 120)

7 Reserpine patient that is can be out of bed

8 Sedation Phenobarbital (Phenobarbital) 0.1 Gm (1 1/2 gr) immediate ly in y r p to 0.03 Gm (1/2 g) q 1 d

# DRUGS USED IN THE TREATMENT OF BRONCHIAL ASTHMA

Preparation	Dose	Mode of Administration and Indication
Epinephrine Injection U S P Injection of Adrenaline B P (1:1000 dilution of the hydrochloride in aqueous solution)	0.2-1.0 cc (3-15 $\mu$ ) may repeat up to q 30 minutes if necessary 1 cc (15 $\mu$ ) in liter of 5% glucose solution Give at 60-80 drops per minute	Subcut. This is the most commonly used preparation 1 V Caution Reserved for very severe acute attacks when more conservative measures fail
Epinephrine in Oil Injection U S P (1:500 dilution)	0.2-1.0 cc (3-15 $\mu$ ) May repeat in 10-14 hours Duration of action is 3-24 hours	Subcut. or I M usually given with aqueous epinephrine to patients with severe or recurrent asthma
Epinephrine Inhalation U S P (1:100 dilution in aqueous solution) Isopropylarterenol (Isuprel <sup>®</sup> ) (1:100 dilution in aqueous solution) For inhalation only	0.5 cc in nebulizer Individualize dose 4-8 inhalations usually suffice (Isopropylarterenol causes less vasoconstrictor action)	Glass nebulizer operated by hand bulb or pressure from an oxygen tank or nebulizer with intermittent positive pressure breathing (IPPB) (see page 149) Most useful in aborting attacks
Isopropylarterenol tablets (3-10-15 mg) Corticotropin (ACTH)	Sublingual individualize dose 10-40 mg I V in 24 hours or 25 mg of regular or gel I M every 8 hours initially	May be useful in aborting attacks Decrease to tolerance in prolonged use Used for severe attacks and status asthmaticus
Cortisone Aminophylline Injection U S P B P (Theophylline Ethylenediamine)	25-75 mg q 6 hours 0.24-0.48 Gm (3-3/4 to 7 1/2 gr) in 10 or 20 cc saline May repeat in 3-4 hours Duration of action 1-3 hours	Orally I V slowly May be used with or without epinephrine Valuable in severe attacks when patient is pinphrine fast
Aminophyllin U S P rectal suppository Ephedrine Hydrochloride U S P B P or Ephedrine Sulfate U S P (capsule or pill)	1 suppository every 4 hours 25-50 mg (3/8-3/4 gr) very 3-6 hours May combine with phenobarbital 15 mg (1/4 to 1/2 gr) or pentobarbital sodium 30 mg (1/2 gr)	Used only when prolonged effect is desired Orally Of little or no use in acute attack May be of some value in aborting an attack or decreasing the number of attacks
Antihistaminic drugs (see page 68) (pill or capsule)	50-100 mg b i d to q i d	Orally Of little use in acute attacks May be of value in aborting attack & decreasing number of attacks

B 5 Atta k Epinephrine responsive p t e ts (may also fol  
low as f r status sthm ti b low)

- 1 Epin phri e inje tion inje ction of adrenaline 0.5 1.0 cc  
(8 m v) 1 1000 oil tion subcut and rep t ev ry 30 60  
mi ut s if nec ssary
- 2 Epinephrine inh l tion 1 100 by n buliz = usi g = yg n for  
ap y m y gi d am i relief May p t ev y 30 60  
min tes
- 3 Epin ph ine in oil 1 500 0.2 1.0 cc (3 15 m) I M f pro  
long d ff t m y help prevent re rr nce of attack a d  
an be repeat d in 10 14 ho rs
- 4 Amin phylline (theophyll thyl edi mi e) 0.24 0.48 Gm  
3 3/4 7 1/2 gr ) in 10 20 cc (2 1/2 5 d ) li e lowly I V if  
atta k not o t oil d M y also gi e th s a ct i instilla  
tion or t l uppository in the ame d se n p m m d n  
5 S d tion must b dequ te Us e of the f llow g  
a P atch bit l sod um (p tob rbt odi m) 0.1 Gm  
(1 1/2 g ) at on e and m y ep t  
b P ald hyde 4 8 (1 2 d ) lly in f ut juice o  
r t lly in 30 e (1 ) l
- 6 100% oxygen (or 20% helium w th 80% oxyg n) inhalet by  
m sk t 8 12 l t s/min te may gi great l ef from  
dy p ea
- 7 Wh n labl the u of oxyg n by it mite t p itiv  
B ur l g B nnett v l e) and b onchod l tng s osia  
dmin t d mulla usly th ough the ame appa stus af  
fo d the m t d am t c r l ef cute ti ks of sthm  
As a bron h d l tor flupropyl sterenal (Isup el<sup>®</sup>) is to be  
preferred b cause it prod ces a l ssar deg ee of syst mic  
r ti then do s epinephrine
- 8 Th pl of rfa etens low ing ag nts ( g Alavair<sup>®</sup>)  
dep lyme ing ym ( g hyal ond ) o digesti  
e yme (e g tryp i ) by ero l in thi e dition is t l  
t d i rmin d Th d is bility (using the l tter h e  
c tly be n qu tion d in v ew of its ffecs on th m rph logy  
f the ll f th sp t r y lining s w ll as t i l t t g  
l cal ff t

C St t a Asthm t d S e Att ch i Ep ph in sta t  
P t t

- 1 C t i ot pin (ACTH) 25 50 mg of r gul r o g i pr p  
a stio b t or l M o ort 25 75 mg orally  
Immed t ly a dr p t y 6 h u s l f th p t t l  
ho p t li d d rami t ti ot p (ACTH) 25 40 mg  
in I V d p er 8 12 hou pe de y 24 hours ACTH  
m y h mo p d t of t on but otherwis both  
e ab t qually ffec t e Rel f hould b e l d nt in 6 12  
h ur and most mpt t fr d m f m th n g man  
f t t ons 24 48 hou s Th m d c t on h o l d p b bly  
b t d f r 7 10 d y s ing d ally d min h a g d s  
aft th f t 4 5 d y s
- 2 Patie t h uld b ho p t l d f po bl in n lle ge  
f r m
- 3 100% o yge r 20% h i lum w th 80% oxyg should b g v n  
by m sk f l f f dy p
- 4 Am ophyllin (th phylline thyl d am i ) 0.24 0.48 Gm

(3 3/4 7 1/2 gr) in 10 20 cc (2 1/2 5 dr) saline slowly I V and by rectal suppository for immediate relief of symptoms. 0 III Gm (7 1/2 gr) may be added to 500 1000 cc of saline and given by I V drip.

- 5 Sedation must be adequate until relief is obtained. Use one of the following:
  - a Pentobarbital sodium (pentobarbitone sodium) 0 1 0 2 Gm (1 1/2 3 gr)
  - b Paraldehyde 8 15 cc (2 4 dr) in 30 cc (1 oz) oil by rectum
- 6 Surface tension lowering agents (Alevaire®) by aerosol may be helpful in some cases (see page 154).
- 7 If corticotropin (ACTH) or cortisone are not available:
  - a As soon as epinephrine responsiveness returns use epinephrine as above. Epinephrine may be administered cautiously 1 cc 1:1000 solution in 1 liter of 5% glucose by intravenous drip (60 80 drops per minute).
  - ✓ b General anesthetic agents may be life saving.
    - (1) Rectal instillation of 30 80 cc (1 3 oz) of ether in equal quantities of olive oil repeat in 12 to 24 hours if necessary. Usually patient awakens free of attack.
    - (2) If a trained anesthetist is available inhalation ether anesthesia may be employed.
- 8 Bronchoscopy under general anesthesia is sometimes indicated to remove tenacious secretions.

#### D General Measures

- 1 Eliminate any known allergens from patient's environment.
- 2 Maintain adequate rest and relieve apprehension by reassurance and sedation.
- 3 Respiratory infections must be treated vigorously with antibiotics as indicated I M or by aerosol.
- 4 Fluids orally or parenterally for any dehydration.

#### Interim Therapy

A Specific Therapy. Attempt to determine which allergens play a role and treat accordingly.

#### B General Measures

- 1 Emotional disturbances should be corrected whenever possible.
- 2 Good living hygiene should be promoted.
- 3 Patients with apparently intrinsic asthma (usually due to infections of bronchi) may be helped by a tibiotic therapy (see pages 115 and 121).
- 4 Ephedrine hydrochloride or sulfate III 50 mg (3/8 3/4 gr) with or without phenobarbital (phenobarbitone) 15 30 mg (1/4 1/2 gr) every 3 6 hours may prevent or reduce recurrences.

#### 5 Aminophylline ephedrine phenobarbital capsules

R Aminophylline	0 2	gr	iii
Ephedrine hydrochloride or sulfate	0 0 5	gr	3/8
Phenobarbital	0 0 15	gr	1/4

Sig 1 capsule every 4 hours

- 6 The new antihistaminic agents may give relief in some patients but their use in bronchial asthma has generally been quite disappointing (see page 66).

Patients who are not helped by other measures may benefit especially with small doses of corticosteroids (ACTH) or cortisone. The dosage employed is just sufficient to keep them comfortable and relatively free of symptoms.

# BRONCHIECTASIS (code No 350 100 6)

Bronchiectasis is a chronic progressive disease of the bronchi and bronchioles characterized by dilatation of the bronchi or bronchioles in presence of varying amount of infective infection and finally destruction of the involved parts and of the surrounding tissue. The etiology in many cases is unknown but chronic focal and chronic or recurrent pulmonary infections undoubtedly play a role.

## Diagnosis

A History

- 1 Chronic cough usually productive of mucopurulent sputum and marked upnaing in the morning
- 2 Recurrent attack of pulmonary infection with aggravated cough fever and chill Hemoptysis is common

## B Physical Examination

Chest finding: Hemoptysis is common during acute pneumonia or erythema. Rales at lung bases and coarse wheezes are typical of the moist rales at mon findings.

## C Laboratory Findings

- 1 X-ray Routine chest x-ray usually insufficient to make diagnosis. If chest x-ray is negative and bronchiectasis is suspected further studies are necessary.
- a Bronchoscopies examination
- b Bronchograms (x-rays of chest following instillation of iodized oil into bronchi either through bronchoscope directly into the trachea) are most important for diagnosis. The study must be made by an experienced radiologist.

- 2 Sputum The sputum is usually found to part into 3 layers: Bacterial, tuberculous and polyvalent infection usually with septum and phycocephaloid microorganisms.

## Treatment

### A Specific Measures

Treatment with antibiotics. It has been found that in temporally limiting symptoms especially during the acute exacerbation both had no effect. The amount of sputum and weight reduced and the patient felt better but the benefit was not sufficient to make the treatment should be repeated since many of the patients would be regarded as cured. When possible predominant organisms should be identified and their sensitivity to the various antibiotics determined. Penicillin, streptomycin, p-aminosalicylic acid, 50,000-100,000 units of penicillin, streptomycin 154 (100 mg) 50,000-100,000 units of streptomycin, penicillin G in 1020 cc, aim at the patient. Penicillin use of penicillin except during attack.



pneumonitis appears to be of less value than direct inhalations

- 3 Streptomycin aerosol (see page 154) may also be of benefit in some patients especially those in whom penicillin resistance occurs. Each cc should contain 50-250 mg of streptomycin depending on concentrations desired. Administer in the same manner as for penicillin (see above).
- 4 Combined penicillin streptomycin aerosols may be of benefit in many cases. Use the same concentrations for the drugs as used individually.
- 5 Oxytetracycline (Terramycin®) is also available for aerosol administration (50 mg/cc in propylene glycol) and may be used as above. (See page 154).
- 6 Mucolytics by aerosol may be of value in obtaining better drainage of thick inspissated material (see page 154).

#### B. C. Management

- 1 Postural drainage. Postural drainage has proved to be the most effective angle measure for the symptomatic relief of patients with bronchiectasis. The patient should assume the position that gives him the maximum drainage and this varies with the location of the lesion. Experience will help the patient determine the best position to use. Since most lesions are in the lung bases the most common method is to have patient kneel on a chair, place hands on the floor and keep hips elevated maintaining this position for 10-15 minutes. Two to four times a day is usually sufficient the first drainage being just upon awakening and the last just before bedtime.
- 2 Avoidance of upper respiratory infections is very important in controlling the bronchial infection.
- 3 Correction of associated disease. Many patients with bronchiectasis suffer from chronic upper respiratory infections with postnasal drip. This must be corrected whenever possible.
- 4 Climate. Although climate does not cure a warm dry climate often is of benefit especially since it tends to reduce the incidence of upper respiratory infections. Avoid a dusty smoky filled atmosphere.
- 5 Rest. Patients with severe disease should always have adequate rest in bed. For symptoms are often ameliorated by this measure. The foot of the bed should be raised 6 to 12 inches.
- 6 Good nutrition and health are very important. Adequate food and rest will aid in slowing the progress of the disease. Smoking should be prohibited.
- 7 Bronchoscopic drainage is of value initially in all cases to eliminate bronchial stenosis or obstruction as to contributing factors. It may be necessary to dilate the stenosed bronchus but repeated bronchoscopy is not advised.

#### C. Surgical Treatment. Properly accepted indications include

- 1 Younger patient in good condition who are having chronic or recurring symptoms of any degree. Modern surgery will permit resection of fairly extensive bilateral disease.
- 2 Patients up to 50 years of age who are having severe symptoms

(espe lly r cu e t h m o r h g ) f m p e d m i n a u t l y u  
l t e a l d s e a a n d w h o a r e o t h w i s g o o d s u r g l r k s

## DISEASES OF THE LUNGS

### PNEUMONIAS

P u m o n i a m f n f l m m t y h a n g s f t h e p a h y m  
f t h e l u g l m a t f w y s t d w i t h o d t o i n f t f  
t h p a t t w c u t m y t l a l y t h p n u m o n i a i n t a t o m l  
t y p e s ( i l b d b n h l ) b t h s l a f t n i t i  
f u l p u p o a t p r n t l n i w f t h v r g o w g n m b o f  
t m b a i g n a e s h u d l l y t h p n u m o o t h  
b s f t l g y h i a t w i t h a p p m i g t  
T h p n u m o e a t l l t h m o t o m m s o f p n m o n  
l n g l i m a y b a t d t h t h m a n g m i f l l t h p u  
m o n i f o l l w t h m p c p l t h o g n a n g t h r a n g  
m n t o f p m l p n m n

### PNEUMOCOCCAL PNEUMONIA (code No 360 101)

#### D i g n o s i s

- A H a t y U s l l y s d d a t w i t h h i l a n d f O f t n  
p i t p n i e e n t w i t h u g h
- B P h y a l F d g s V y w i t h t t l o c t m n d d t n f  
t h p a i r i g l p o c f m e n f w i n e r l t m  
a t e n s i d i t o n f n e o m o i b
- C L a b t y  
1 S p u m P o l i u a l l y t g d w t h b l o d ( l i g h t p k t  
t h e o l f p u e j )  
2 A l l p t i w i t h p m o ( p l l y f ) h i d h  
t h f l l w g i b a t y x m t d d t n t u t  
u a n d b l o d t d i s h t a y p t m e n t ( p m  
e t p r l m m n f i d ) d b l a d i t

#### T r e a t m e n t

B f o r b g a n u n g t h p y t d a b i t h t m t m a n d  
b l o o d f i t d i d i m n e t h e t b t i a n d  
T h a u m p s f i t h u n t o m e  
T h r o n p w a r t w i t h t h e s e w r i t s f t h e d i a d e  
f e r m i n e t h e s e x i s p r o i n g s i n a n t r a h a m i n  
t h e o p a t n a e i b a d r e a t a u t i h d b l o w

#### S p e c i m e n s

P n l i t h d g f h m p m o c l i n f t  
C h i o l t y l u x y t t y l a t t y l l a l o r n e p h  
a n d e r y t h o m y n a r i s o h i g h l y f i t m m t p m o c c  
i n f t o n s b t a p r o b l y l i g h t l y i n f a t p m l l a n  
i n f t o n s T h s i f n m d d g a a i s o f f i b u i t h  
p n e t o p i l i m a l l y m p d n d o m p l t h n  
l a f q u t

## Therapy Based Upon Evaluation of Factors Influencing Prognosis

Factor	Mild or Moderate	Severe	Very Severe
Age	Under 40	Over 40	
Organism count in sputum/oil field	Under 30	30-75	Over 75
Lobes involved	Single	2 or 3	4 or 5
Pulse rate	Under 120	Over 120	Over 140
Blood pressure			Shock or pulmonary edema
Systolic	Over 90	Under 90	
Diastolic	Over 60	Under 60	
Leukocyte count	Over 10,000	8,000-10,000	Under 6,000
Albumin RI	0 to ++	+++	
Associated disease	0 to mild	Moderate	Severe
Complications	None or sterile effusion	Empyema lung abscess etc	Myocarditis and endocarditis
Blood culture	Negative	Positive	
Pneumococci type	Higher types	I II III IV VII	
Mortality rates			
Range	0-10%	25-10%	10-50%
Average	0-4%	4%	20%
Therapy indicated	Usual doses of penicillin broad spectrum antibiotic or sulfonamide	High doses of penicillin broad spectrum antibiotics or sulfonamide	Massive doses of penicillin

(Modified from Miller & F. Collen, Ferman, the Foundation Medical Bulletin VI 31, January 1948)

may be administered in several ways. The soft rubber facial mask of the BLB, OEM, or Bennett type is probably best. With these masks oxygen concentrations up to 95% may be easily furnished. Oxygen tents are generally used for patients in toxic delirium who would otherwise remove the mask. However, the tent is generally not advised because the average concentration of oxygen is only about 40-50%, and unless watched carefully carbon dioxide may accumulate.

- B. Fluid. Fluid intake must be adequate whether given orally or parenterally to maintain a urine output of at least 1500 cc. Patients taking sulfonamides should have sufficient alkalizing powders so that the urine is basic pH 7 at all times. Potassium bicarbonate should be used in patients with actual or potential heart failure, care being exercised to avoid potassium toxicity.

- C. Diet. During the severe acute phase patients usually have little desire for food. During this short acute phase food intake is of little importance. Patients who develop complications and have long convalescences should be placed on high protein, high vitamin, high calorie diets.

## Symptomatic and Supportive Measures

A. Toxic Delirium. The intensity of the toxic delirium which may occur in severe pneumonia must be controlled



from the intestines

- 2 Neostigmine Methylsulfate U S M (1 2000 Sol ) 1 cc (15 m) subcut and insertion of a rectal tube will generally produce rapid initial decompression
- 3 Stomach tube for dilatation of the stomach Suction through a nasal tube passed into the stomach is necessary

#### F Cardiac Abnormalities

- 1 Congestive failure In elderly patients or patients with pre existing heart disease congestive failure may be precipitated by the pneumonia When this occurs digitalization by one of the rapid methods is indicated (see page 197) This must be distinguished from shock and pulmonary edema (see page 127)
- 2 Cardiac arrhythmias The occurrence of extrasystoles is common and generally requires no treatment If auricular fibrillation or flutter develops rapid failure may be precipitated Digitalization by one of the rapid methods is generally indicated in these cases (see page 197)

#### Complications

For treatment of these complications see the respective diseases (Modified after Collen )

Complication	% Incidence
Sterile pleural effusion	4 5
Empyema	0 3
Lung abscess	0 3
Pericarditis	0 3
Endocarditis	8 1
Meningitis	0 1

All pleural effusions associated with pneumonia must be aspirated promptly to detect early empyemas which may be treated medically (see page 142)

#### STREPTOCOCCIC PNEUMONIA

(Lobar code No 360 10\*) (Bronchopneumonia code No 361 102)

An uncommon type of pneumonia usually secondary to a preceding pulmonary infection (i e virus pneumonia influenza or measles) Onset is most often gradual but is at times sudden with severe intoxication marked dyspnea and cough with bloody or mucopurulent sputum Pleural effusion occurs early is fairly common and may progress to empyema Most cases are due to  $\beta$  hemolytic streptococci

Physical findings vary with severity there may be only scattered dullness and moist rales In severe cases pleural effusion obscures pulmonary signs Throat is usually reddened and has some exudate

#### Treatment

Penicillin is the drug of choice Dosage is similar to that for pneumococcal pneumonia

# STAPHYLOCOCCAL PNEUMONIA (code No 361 105)

129

An uncommon type of pneumonia usually secondary to a preceding infection. Onset is most often gradual and progressively with patient becoming gravely ill. Cough and dyspnea are common. Multiple lung abscesses occur frequently. Patchy consolidation with diffuse consolidation commonly found. Sputum is variable in appearance.

## Treatment

Sensitivity tests vary greatly in their response to penicillin and other antibiotics. Sensitivity tests should be performed. Meanwhile therapy should be initiated with erythromycin in 0.5 Gm every 6 hours pending result of the tests.

# FRIEDLANDER'S PNEUMONIA (code No 361 131)

Pneumonia due to *Klebsiella pneumoniae* is often associated with chronic debilitating diseases. The onset is usually sudden with chills, fever, dyspnea, cyanosis, cough, and marked toxicity. In most cases progress is rapidly to a fatal termination. There is a tendency to necrosis and abscess formation in the affected or chronic form. Early recognition is imperative for favorable outcome.

Physical findings are variable and extensive in old patients. Give only dullness and diminished breath sounds. Sputum is reddish mucoid and tenacious giving a currant jelly appearance. White blood cell count is variable. May have leukopenia or leukocytosis.

## Treatment

Specific Measures Treat all Friedlander's pneumonia as severe infection.  
1. Streptomycin, 1 Gm every 6 hours until if possible response occurs then 0.5 Gm every 6 hours until after 3 days.  
and 2. Oxytetracycline (1 M o l V) chlor tetracycline or tetracycline or chloramphenicol 0.5 Gm every 6 hours or Gantrisin® 1 Gm every 6 hours. Continue for 2-3 weeks.  
B G n a l M See Pneumococci Pneumonia page 123.

# HEMOPHILUS INFLUENZAE PNEUMONIA (code No 361 110)

A rare form of pneumonia which usually is rapid in onset and progresses. The outstanding feature is severe inflammation of the bronchi and bronchioles leading to bronchitis and hemorrhagic drainage of lungs. Patients are toxic and may die. The patient is a pathologically old individual. The sputum is bloody. Leukopenia is frequently present.

## Treatment

Specific Measures Continue treatment for 7-10 days if response is not evident.  
1. Combination of streptomycin and erythromycin if available. Streptomycin 0.5-1 Gm every 6 hours.

has been complete clearing of the lung by x ray. Serial x rays are required in this follow up which may take many weeks.

### NEOPLASMS OF THE LUNGS BRONCHOGENIC CARCINOMA (code No 350 8 ) (Epidermoid: code No 350 814)

Neoplasms of the lungs form a very important group of the malignancies. The metastatic tumors are most common but the primary neoplasms are of great interest in diagnosis and therapy. The primary ones usually arise from the bronchi and spread into the lung fields. They are rarely diagnosed early because of the insidious onset and tendency to mimic other pulmonary disease. Chronic cough is the common presenting symptom. As the process spreads the cough becomes productive and hemoptysis, consolidation, atelectasis, lung abscess and pleural effusion may occur. It must always be considered in the diagnosis of any acute or chronic pulmonary disease, especially in males over 50 years of age. Bronchoscopy and examination of sputum for cancer cells are important diagnostic tools.

#### Treatment

- A. Surgery is the treatment of choice when the lesion is discovered early.
- B. Supportive and symptomatic measures for those cases in which surgery cannot be performed.

### PULMONARY ATELECTASIS (Compression: code No 362-435) (Postoperative: code No 362-415 4)

Atelectasis is due to obstruction of a bronchus with absorption of the air and collapse of the lung distal to the obstruction. Most cases follow major surgery and tend to occur in the right lower lobe. The condition usually is manifest 1-4 days after surgery and the findings are those of poor ventilation and collapse of the involved area. If immediate treatment is not carried out, secondary bacterial infection occurs and a pneumonitis develops.

#### Treatment

##### A. Postoperative Atelectasis

1. Force patient to hyperventilate either voluntarily or by use of a mixture of 95% O<sub>2</sub> and 5% CO<sub>2</sub> administered by mask for several minutes every 1-3 hours. This is also a good preventive measure.
2. Bronchodilatation by aerosol with intermittent positive pressure (e.g. Bennett) has been demonstrated to resolve many cases of postoperative atelectasis. The apparatus should be used every 30 minutes out of every hour while patient is awake for 24 hours before deciding whether further treatment is of value.
3. If rapid resolution does not occur, the mucus plug should be aspirated through a bronchoscope.

4 Institute antibiotic therapy P ocaine penicillin complex  
300 000 unit b i d  
B Spot neo s At le t size B onchoscopy to determine the nature  
ith bair t on and th n institute pprop late tr atment

**PULMONARY EMPHYSEMA**  
(Due to Unknown Cause code No 362 9x8)  
(Postural code No 362-434)

Pulmonary emphysema is a disease usually found in older individuals and the suffering from chronic bronchitis and asthma. The progress is a continuation of the pulmonary infection with subsequent development of the interlobular septa and peripheral emphysema. The diagnosis is generally not difficult. The chief complaint is dyspnea. The anterior-posterior diameter of the chest is usually increased. The lung fields are hyperinflated and breath sounds are decreased. Pulmonary emphysema must be differentiated from dyspnea due to congestive failure.

T t m s  
A Sp i M a u e s  
In many patients have associated chronic bronchitis and elements of psammoma bodies. The clinical picture is similar to that of chronic bronchitis or asthma (see page 117).  
1 Spasmolytic agents to relieve bronchospasm. (see page 118)  
2 Educate the patient to use the inhaler properly. (see page 118)  
B C I M b e  
Inhalation of 100% oxygen for 20-30 minutes helps relieve dyspnea. The patient should wear an abdominal belt during the treatment. The patient should wear an abdominal belt during the treatment. The patient should wear an abdominal belt during the treatment.

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The patient should wear an abdominal belt during the treatment. The patient should wear an abdominal belt during the treatment. The patient should wear an abdominal belt during the treatment.



- 2 Inje 1 50 000 100 000 units of penicillin in 10 cc saline into the pleural space through the same needle This is for either prophylaxis or actual therapy

### B Pleural Fluid Examination

- 1 Gross examination Take specific gravity to determine if exudate or transudate  
Smear and stain for detection of organisms and nature of cellular content Collect a specimen in an anticoagulant for cell count
- 2 Culture on appropriate media and inoculate guinea pig with all fluids from unexplained pleural effusions to rule out presence of tubercle bacilli or fungi
- 3 Pathological examination of centrifuged button In suspected cases of malignancy

### Treatment of Post-pneumonic and Other Sterile Effusions

- A Specific Measures All prophylactic measures are directed at the primary disease Begin or continue antibiotics in dosage used for treatment of pneumonia (see page 123) until patient has been afebrile for 10-14 days or fluid has almost entirely resorbed

### B General Measures

- 1 Thoracentesis Whenever a chest tap is performed instill 50 000 100 000 units of penicillin in sterile saline
  - a Remove readily obtainable fluid by multiple thoracentesis at daily intervals if necessary Removal of more than 1000 cc at a time is not advisable
  - b Re-examine pleural fluid to rule out empyema if the pleurisy does not respond to treatment
- 2 Bed rest until patient is afebrile

### Treatment of Tuberculous Effusion (code No 370 123 B)

- A Specific Measures Treatment for uncomplicated primary effusions is essentially the same as that for minimal pulmonary tuberculosis A course of streptomycin and PAS is recommended (see page 135)

### B General Measures As for pulmonary tuberculosis (see page 131)

- 1 Bed rest most important
- 2 Thoracentesis Removal of all readily obtainable fluid (see above) is advisable to minimize later thickened pleura
- 3 Pneumoperitoneum is used by some for the underlying lesion It is initiated after fever has subsided and active fluid formation has stopped
- 4 When high fever persists longer than two weeks hematogenous dissemination should be suspected

- C Follow-up Treatment Careful follow-up for a 5 year period is necessary because many patients with primary tuberculous effusions develop pulmonary tuberculosis later usually within 5 years

### **EMPYEMA (code No 370 100)**

Empyema is usually secondary to pulmonary infection but may result from direct contamination of the pleural space through trauma

o thoracic auscultation The patient is usually quite ill and the cough is  
frequent and severe

### Treatment

- A Specific Measures System administration in high dosage (100 mg per 4 hr) in therapeutic or antibiotic agent as determined by examination of infecting organism. Treatment should be continued for 10 to 14 days after patient is afebrile and fluid has become sterile (see page 514)
- B Daily hydrothorax should be performed if necessary as a result of the pleuritic pain is possible. Frequent physical examination of the chest with x-ray must be done to avoid overlooking any loculated areas (pocket) of purulent material.
  1. If significant pleural effusion with 500-2000 cc a liter
  2. If at least 500-2000 units of penicillin and 500-1000 units of streptomycin in 10-15 cc saline to the cavity with completion of the irrigation. Continue daily until fluid in cavity has been sterile for 10-14 days or until fluid is no longer obtainable
  3. Various enzymatic agents have recently been prepared which digest protein material especially fibrin to form in this condition. These are being introduced directly to the thoracic cavity. The preparation contains a mixture of trypsin (Trypsin®) and streptokinase (Streptokinase®) (Vidase®). Trypsin is hydrolyzing fibrin into polypeptides. The streptokinase is a streptococcal enzyme which attacks only fibrin.
- C Steroid hormone must be administered if pleural effusion improves with a few days.

### HYDROTHORAX (code No 370 522)

Hydrothorax is most generally due to congestive cardiac failure. Treatment should be directed to the cause of congestive failure and removal of the fluid is necessary.

### HEMOTHORAX (code No 370 532)

If method of examination is general, it is to be used. World War II experience has shown that separation of the blood from the pleural cavity is the treatment of choice. Repeated aspirations are preferred as surgery is not indicated in the case of patients who develop blood after laceration of the pleural cavity. The pleural cavity is not a closed space but a space which is constantly being renewed; therefore, the condition is not a closed space.

### PNEUMOTHORAX

A. In the pleural space, occurrence of air enters the thoracic cavity through the chest wall (by artificial pneumothorax or rupture) or as a result of air entering from the lung.

## HYPOXIA. COMMON CAUSES AND METHODS OF CORRECTION

Physiological Classification	Clinical Condition	Treatment
Hypoxia With Normal Lung Diffusion in atmosphere	High altitude flying	Oxygen at atmospheric pressure
	Cyanosis (not poisoning)	Respiration treatment
Airway obstruction	Poisoning	Cyanosis
	Aspirated food in trachea of larynx and pharynx glottis in trachea	Oxygenation by nasal cannula
Pulmonary embolism	Asthma	Respiration
Pulmonary edema	Emphysema	Respiration
Hypoxia With Abnormal Lung Diffusion in atmosphere	Pneumonia	Oxygen at atmospheric pressure
	Emphysema	Oxygen by nasal cannula
	Chronic bronchitis	Oxygen by nasal cannula
	Chronic bronchitis	Oxygen by nasal cannula
Impaired diffusion in pulmonary capillaries	Pulmonary edema	Oxygen by nasal cannula positive pressure (in trachea)
Various Artistic	Cyanosis	Oxygen at atmospheric pressure
Hypoxia Due to Impaired Oxygen Transport by Blood	Anemia	Cyanosis
	CO poisoning	Oxygen at atmospheric pressure
	Congestive heart failure	Oxygen at atmospheric pressure
	Shock	Oxygen at atmospheric pressure
Hypoxia Due to Impaired Oxygenation	Edema	Oxygen at atmospheric pressure

ported in incidence of oxygen therapy have been established on the basis of results from properly humidified oxygen.

## TECHNIC OF ADMINISTRATION

Oxygen (air) may be administered at atmospheric pressure or by various pressure devices.

### OXYGEN AT ATMOSPHERIC PRESSURE

Oxygen is most commonly administered at atmospheric pressure. It is indicated when hypoxia can be controlled adequately by the usual means. For indications see page 146.

Various methods of determining oxygen at atmospheric pressure are available. Below are listed those methods which are most commonly used with adult and the oxygen concentration which can be achieved.

Method	Usual Oxygen Concentrations	Usual Rate of Oxygen Flow (L/min)
Tent	40-50%	Flow 15-20 Minimum 12-15
Canister		
Nasopharyngeal (nasal cannula)	20-40%	6-8
Oronary cannula	30-40%	6-8
Mask		
BLB or simple mask	80-100%	8-10
Exp. dil. plastic mask	40-60%	10-1
OEM or Bennett		
face mask	80-100%	6-8

### Oxygen Therapy

#### A. Administration

1. Give moderate to full flow of oxygen at minimum concentration to the patient.
2. Can be used with stillness and uncooperative patient.

#### B. Devices

1. Most effective therapy and practical.
2. Cannot be effective if flow of oxygen is not properly adjusted to the patient's needs.
3. If not properly adjusted, it may lead to hypoxia.

#### Nasopharyngeal

This is the apparatus for oxygen administration consisting of a small catheter (Fr. No. 10 or No. 12) with the terminal hole positioned by a small bulb and a suction valve mechanism and a humidifier bottle.

#### A. Technique

1. The tube should be lubricated with petrolatum and placed every 6-12 hours.

## 148 Oxygen Therapy

- 2 It may be placed in the nasopharynx or 1 2 inches into nares but concentrations are only up to 20% by this method
- 3 Place in oropharynx for concentrations up to 40% To calculate the approximate distance the tube must be inserted measure the distance from the external nares to the tip of one ear lobe using the tube to measure with Then pass the tube through the nose into the oropharynx When the patient begins to swallow withdraw the tube about  $\frac{1}{2}$  inch and secure it in position

### B Advantages

- 1 Cheapest method of administering oxygen
- 2 Patient is less uncomfortable than with mask

### C Disadvantages

- 1 Very high concentrations of oxygen are not obtainable
- 2 Drying of mucosa may result with ordinary humidification

## Masks

### A Apparatuses

- 1 BLB masks Nasal or oronasal rubber mask with rebreathing bag The disadvantage of this mask is that with low flow of oxygen (under 6-8 liters per minute)  $\text{CO}_2$  tends to accumulate There may also be resistance to inspiration from flat rebreathing bag
- 2 Expendable plastic masks Require high oxygen flow Low oxygen concentration achieved
- 3 OEM and Bennett face masks Similar to BLB mask but do not permit rebreathing into bag utilizes flutter type valve so rebreathing of  $\text{CO}_2$  is not possible

### B Advantages of Masks

- 1 Highest concentrations of oxygen obtainable without the use of pressure (except for plastic mask)
- 2 Both OEM and Bennett masks have injector settings so that oxygen concentration can be varied from 50 to 100%

### C Disadvantages Tight fitting masks cannot be tolerated by some patients

## OXYGEN UNDER PRESSURE

Various pressure breathing devices have been developed which allow oxygen to be administered under slight positive pressure during the inspiratory phase Although originally these devices were employed for resuscitation (usually with a negative pressure phase in expiration) the value of intermittent positive pressure in the treatment of various acute and chronic pulmonary and cardiac conditions was soon recognized

### Physiological Effects

The principal physiological effects of oxygen administered by pressure methods are as follows

- 1 Helps overcome resistance to gas flow and widens the bronchioles permitting more efficient cough and bronchial drainage
- 2 Increases intrapulmonary mixing creating more uniform alveolar aeration

- 3 Discreases residual volume
- 4 Inhibits fluid extravasation into the alveoli (phenomenon of alveolar pulmonary edema)
- 5 Interferes with venous return to the right heart with consequent decrease in cardiac output and blood supply to the lungs. This latter phenomenon is of value in management of congestive failure especially with associated pulmonary edema in shock. On the other hand it is a disadvantage and often contra-indicates the use of positive pressure device in this condition.

### PRINCIPAL METHODS OF POSITIVE PRESSURE BREATHING

Method	Pressure During Inspiration	Indications and Uses	Remarks
Mouth to mouth or mouth to endotracheal tube	Positive pressure in inspiration	Mainly used in children and newborn infants	Most primitive method of positive pressure but may be very effective. Oxygen administration at lower than atmospheric concentration
Bennett positive pressure therapy unit (motor or oxygen powered)	Positive pressure in inspiration. May use oxygen, air or oxygen-helium mixture	Mainly for therapy of chronic pulmonary diseases. Also useful in pulmonary edema	Interferes with venous return to right heart so contraindicated in forward failure. Especially useful in hypoxia due to improper mixing of gases and for pushing oxygen across impaired membranes (see page 150)
Oxygen jetor mask (Bach) mounted for positive pressure	Positive pressure in inspiration. Used with oxygen	Adapted to pulmonary edema	Letting in of Positive pressure applied at wrong place in respiratory cycle to be of benefit. Also very tiring to breathe against resistance
Commercial resuscitators of the Salk and blow type. Stephens, Ferson, E and J etc.	Positive pressure in inspiration and negative pressure in expiration. Generally employ oxygen	Resuscitation	Most effective means of resuscitation. Least interferes with cardiovascular dynamics although the usual pressure relationship in inspiration and expiration is reversed. Negative pressure may cause pulmonary edema in predisposing conditions
Hand bellows	Positive pressure in inspiration	Resuscitation	Expensive equipment but useful mainly when more expensive apparatuses are not available

Bennett Positive Pressure Therapy Unit

The Bennett unit is one of the most efficient of the available pressure breathing devices. It may be used with an intermittent nebulizer or with Mist O<sub>2</sub> Gen® continuous nebulizer for good humidification (or for administration of various antibiotics, vasodilators and surface tension lowering agents). It is a particularly useful way to administer aerosols for it drives them down into the terminal bronchioles and alveoli. Excellent instructions are supplied with the unit. A special apparatus is also made that cycles automatically. Clinical indications and uses are as follows.

1. **Bronchial asthma** Especially with a bronchodilator useful mainly in the acute attack
2. **Chronic emphysema** Idiopathic or accompanying fibrosis, pneumoconiosis, etc. Best results apparently when bronchodilators are used. Must be used cautiously or with automatic cycling in patients with severe hypoxia and elevated CO<sub>2</sub> tension. (See dangers of oxygen therapy page 145.) In these conditions therapy must be employed 2-4 times daily for about 20 minutes per treatment. Treatment is given in courses of 5-30 days which may be repeated as indicated.
3. **Bronchiectasis** As for emphysema above. Antibiotics by aerosol are often useful in this condition.
4. **Pulmonary edema** Especially useful when associated with severe anoxia. Must be used with great caution if shock (forward failure) is present.
5. **Irritating gases and fumes** Very valuable especially with any associated pulmonary edema. Use until lungs have cleared.
6. **Atelectasis** See page 138.
7. **Respiratory depression** Must be used with caution if circulatory failure is also present.
8. **Right heart failure** Helps correct hypoxia, relieve a burden on right heart. Excellent in management of acute right heart failure in conjunction with other measures (see page 182).

Caution

The Bennett apparatus must be used with great caution in all cases of peripheral circulatory collapse or shock (forward failure).

## MAINTENANCE OF RESPIRATION BY ALTERATIONS OF CHEST WALL PRESSURES

Although failure of ventilation may be corrected by pressure breathing devices applied to the airway, this method is not always readily available and cannot be employed for long periods of time. In such cases respiration can be maintained by applying pressure variations to the chest wall. When these methods are employed the normal intrapleural pressure relationships are maintained. The usual methods are outlined on the following page.

# PRINCIPAL METHODS OF PRESSURE ALTERATIONS TO THE CHEST WALL.

M th d	Pre u e Du ing Re p iration	Indic tion a d Use	R marks
Artificial respiration	The best method to use is to pull on a m t expand chest f in p i t i o n pre a u e f r x p i t i o n ( e b l o w )	All p tory f i u r e s w h e n o t h e r method is v a l l b l	E f f e c t i v e p h y s i o l o g i c a l w h e n p r o p e r m e t h o d i s u s e d ( s e e p a g e 152)
Body s p i t r	Neg t i v e p e u ( s c t i n ) f o r i n p i t i o n u s u a l l y u d M y u s p a s s i v e p r e s s u r e ( i n s p i r a t i o n ) i n a t t e m p t t o c o m e e p o l u n g	R e s p i r a t o r y f a i l u r e w h e n p o l g d a i d i s e e d e d	S i n e n e g t i v e p r e s s u r e a p p l i e d t o e n t i r e b o d y m y d u m i n i E a r d a f i l l g d u t p o o l i n g o f b l o o d i n e x t r m i t e n d t n k m a y b d a n g e r o u i n f o r w d f a i l u r e
C i s r s p i t o		A f o b o d y r e s p i r a t o r e s p e c i a l l y i n c h i l d r e n e t a b l i d p o l o m y e l i	M e p h y s i o l o g i c a l t h a n b o d y r e s p i r a t o r L s i t r f r n w t h i c l a t i o n O f t o m f r t b l e r h a d t f i t f o p o l o n g d i t i m U n s t a b i l e y i n e l y r e p o l l i o m, i t l
R c k i n g b e d	R e p l i c n t r o l l e d m a i n l y b y a b d o m i n a l c n t e t s d r o p p i n g a w a y f r o m o r p u l l i n g d i p h g m	L e e d g e a o f p r a t o r y f i l a a b	M e m t l l t f i m p o v i n g i u l t o y d y n a m i c a d n a l d r a i n a g i e t i e n t s w i t h m b i d r p i a t o y n d b o d y p a a l y s i s

## ARTIFICIAL RESPIRATION

Artificial respiration should be administered promptly to a person who stops breathing and whether or not drowning suffocates. It is the duty of the first aid attendant to institute artificial respiration should never be postponed while waiting for the arrival of the institution of treatment with a mechanical resuscitator.

This procedure places positive pressure on expiration and provides oxygen to the tissues until the pulmonary pressure is restored. The reverse and eum is a malfunction. A ligament is torn in the chest the pleura has a fracture and the many occur even after many hours of artificial respiration.

The purpose of the methods of artificial respiration is more than to save life as the implanted method (e.g. Schfer)



- ☐ Drugs and Concentrations Employed (Should be prepared fresh daily) The frequency and duration of treatment depends upon the disease and its severity

1 Antibiotics

- a Penicillin Usual dose is 50 000 100 000 units per treatment Dilute in 1 0 2 0 cc of water
- Streptomycin 0 25 0 5 Gm in 1 0 2 0 cc of water
- c Oxytetracycline (Terramycin®) aerosol ■ 100 mg in 1 0 2 0 cc 75% propylene glycol

2 Enzymes

- a Although trypsin (Trypsin®) has been advocated to dissolve thick tenacious mucus or dead tissue in chronic bronchitis or bronchiectasis untoward reactions and bizarre changes in cells have been observed with its use ■ must be used with great caution following instructions carefully  
Dosage 1 3 cc of trypsin solution (40 000 units/cc) prepared by dissolving the dry powder in a special buffer (pH 7 1) Administer 4 times daily for up to 4 5 days for each course Do not employ within one week after frank hemorrhage May be combined with penicillin streptomycin and bronchial dilators
- b Other enzymes have been used (e g deoxyribonuclease (Dornase®) but must still be considered experimental

3 Bronchodilators

- a Isopropylarterenol (Isuprel®) 0 1 0 5 cc of 1 100 or 1 200 solution
- b Epinephrine (Adrenaline) 0 5 cc of 1 100 solution

- 4 Surface tension lowering agents Various surface tension lowering agents have been used to aid in spreading and formation of aerosols Their value is still uncertain Among these drugs are Alcolac® ethyl alcohol etc

D Methods of Administration

- 1 Oral inhalation during inspiratory phase For the greatest effect and efficiency the aerosol should be inhaled through the mouth

- a Continuous pressure from oxygen tank (see lower diagram) A Y tube is inserted between nebulizer and source of pressure Nebulization will occur only when the unattached end of the Y tube is closed by the thumb or a finger there is usually a few seconds delay before the aerosol arrives at the mouth piece

- Intermittent pressure (e g foot bellows or pump) is applied during inspiration

- 2 If the patient is unable to cooperate the nebulizer may be used with an oxygen mask which has a rebreathing bag attached The nebulizer is placed between mask and oxygen

## DISEASES OF THE HEART

C g t i d i s o d f t h i s a t w h i h a m e n a b l e t o a r  
g u e l e t i n a p s t e d b l o w  
A l t h g h m y e s t h d g n o s s f g n i t a l h e a t  
d i a b m d l i n l g r o d a l n m o s t a s e q u i z  
p i l s t d s w h a h e b s t p f m e d n m d l c n t e r s w h  
p o c e d a s d i c c a t h t i t i o n v n o n d r t o g r e d  
t l a g i o g r a p h y e n d t m g r p h y a n t u t a b l f t h o c c a  
s n a l i n v t i g a t e t h y a d i f f u l t t o p r f o m a d i n t e p i d  
E o u s k i l l d i a m w o k

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## 156 Coarctation of Aorta

### Pulmonary Atresia (Code No. 4711 018)

In this variety of tetralogy of Fallot the pulmonary artery is atretic an anastomotic procedure (Blalock) is advised

### Tricuspid Atresia (Code No. 452 017)

This condition is recognized by the combination of a centrally cyanotic congenital heart lesion a dominant a wave in the venous pulse and evidence of left ventricular hypertrophy clinically and electrocardiographically. A Blalock anastomotic operation is the treatment of choice.

### Patent Ductus Arteriosus (Code No. 40x 0x0)

This relatively common lesion varies in severity from complete absence of symptoms to frank cardiac failure.

The indications for ligation of a patent ductus arteriosus in the presence of pulmonary hypertension have not been established. Current opinion favors ligation whenever the flow through the ductus is permanently or intermittently from left to right.

Because of the low operative mortality rate (less than 1%) in skilled hands ligation is recommended in all individuals under the age of 30 perhaps 35. The question of ligation versus division is not settled but the latter has a higher mortality rate. The mortality rate also becomes higher as the patient becomes older. This necessitates caution in recommending surgery in adults who are asymptomatic and have no left ventricular hypertrophy.

### Coarctation of the Aorta (Code No. 461 015)

This condition is characterized by elevation of the systolic blood pressure in the arms but not in the legs a weak delayed femoral pulse as compared to the radial and brachial pulses a systolic murmur heard at the base of the heart anteriorly and posteriorly and signs of collateral circulation circumventing the constricted aorta. If the details of the narrowed aorta can be visualized the diagnosis can be established by retrograde carotid or brachial arteriography or venous angiography.

Resection of the coarcted site is a more formidable operative procedure than ligation of the patent ductus arteriosus and the surgical mortality is in the neighborhood of 5% even in the best hands. For this reason not all physicians recommend routine resection in asymptomatic individuals. The risks of the disease are such however that if a skilled cardiologist surgeon is available all coarctations up to the age of 20 years should be resected. Between the ages of 20 and 35 surgery is advisable if events make it clear that the patient is doing badly.

### Atrial Septal Defect (Code No. 412 0xx)

Surgical correction of atrial septal defect is now possible but the exact surgical procedure of choice has not been established as yet. The results are promising but in view of the limited experience surgical repair should be advised only in those patients showing advancing cardiac failure.

### Anomalous Pulmonary Venous Drainage (Code No. 485 02)

Surgical correction of anomalous pulmonary vein drainage is in the exploratory stage.

## HYPERTENSIVE CARDIOVASCULAR DISEASE

(code No 400 533)

- Hypertension is manifestation a hemodynamic sign and the course of the disease is adversely affected by
- 1 Cardiac failure secondary to increased work of the heart and relative or absolute coronary insufficiency
  - 2 The development of atheromata especially in the cerebral and coronary arteries with syndromes resulting from vascular occlusion
  - 3 Acute vascular necroses resulting from rapid sustained rises of diastolic blood pressure usually exceeding 130 mm Hg which produce the complication known as the malignant phase. Renal failure occurs almost exclusively in this last group

### Evaluation of the Hypertensive Patient

- A Hypertension is a complex of signs and may be present in a variety of diseases, many of which are curable or can be modified by treatment. The first step in the treatment of a patient with elevated diastolic blood pressure is to recognize the probable conditions in which hypertension occurs. These diseases include unilateral renal atrophic kidney often with pyelonephritis, bilateral chronic uropathy, pheochromocytoma, Cushing's disease, visceral angitis, coarctation of the aorta, and acute glomerular nephritis.
- B The assessment of the severity of the vascular hypertension and the integrity of the vital organs commonly affected by hypertension (heart, brain, fundi, kidney) must be determined before therapy can be planned.

### Classification of Severity of Hypertension and Its Complications

Hypertension may be classified as follows:

- A Severe - Patient develops often exudates in the fundus of the eye, disabling dyspnea, disabling coronary insufficiency, epistaxis, cerebral embolism with neurological sequelae, rapidly advancing digitalis hypertension with progressive left ventricular hypertrophy.
- B Moderate - Signs of left ventricular hypertrophy, retinal arteriosclerosis in the fundi, old cerebral embolism with sequelae, slightly or totally coronary insufficiency.
- C Mild - Diastolic blood pressure below 135 with minimal or no objective signs of vascular damage in fundi, heart, brain, kidney.

### Methods Available for Lowering Blood Pressure

- A Drugs (See p 158)
- 1 Rauwolfia compound
  - 2 Veratrum compound
  - 3 Hydralazine hydrochloride (Ap-soline<sup>®</sup>)
  - 4 Mithrium compound
  - 5 Thiocyanate and nitrite
- B Sympathetic nerve
- C Low salt diet
- D Others (e.g. physical therapy, sedation)

Indications for Potent Hypotensive Drugs and/or SympathectomyA Definite Indications

- 1 Malignant hypertension
- 2 Hypertensive cardiac failure when acute myocardial infarction has been excluded (if possible)
- 3 Rapidly advancing diastolic blood pressure with left ventricular hypertrophy and dilatation Evidence of deterioration in the heart and fundi (exudates and hemorrhages) especially in young (particularly male) individuals

B Possible Indications (in Exploratory Stages)

- 1 Recurrent mild cerebral thrombosis with neurological sequelae
- 2 Intractable coronary insufficiency
- 3 Asymptomatic men with diastolic blood pressures between 125 and 130 but without other evidence of complications of hypertension
- 4 Severe intractable hypertensive headaches

C Not Indicated With Present Knowledge

- 1 Mild benign essential hypertension in middle aged women without objective evidence of vascular deterioration or complications
- 2 Early transient hypertension in young individuals without objective evidence of vascular deterioration or complications

Hypotensive Drugs

Many patients with hypertension especially middle aged women live many years in comfort Therefore great care should be exercised and a clear indication of significant decrease in the span of life obtained before subjecting these patients to the disagreeable side effects and potential dangers of a continuous program of drug therapy Hypertension varies strikingly in severity in different patients treatment at present should be varied depending on the severity of the hypertension and the presence of complications

Several drugs are now available but the indications for their use are still not clearly defined Patients with moderate or severe forms of the disease should be given the benefit of a trial of therapy Use the least toxic drugs for mild hypertension Over a period of months or years slight to moderate lowering of the blood pressure may prevent or decrease or possibly reverse the vascular complications of hypertension Combinations of drugs have been used and may prove to be useful but they are difficult to evaluate The addition of rauwolfia is best tolerated since the complications from its use are least

In most severe cases hexamethonium should be considered but in some instances of less severe disease it will be worthwhile to begin with rauwolfia and then either add another drug or change to a more toxic drug if rauwolfia is not effective

A Rauwolfia Drugs Rauwolfia is the most recent addition to the list of hypotensive drugs It has a relatively slight hypotensive action but may be useful because of its mild sedative effect and its value as an adjunct when combined with hexamethonium or Apresolin® It is the least toxic of the hypotensive drugs nasal stuffiness is the most annoying side effect Dose

- 1 Reserpine alkaloid (Serpasil® Reserpidin®) 0.5 mg tid orally

2 Aise oxylon alkaloid (Rautensin®) 2 mg q i d

3 R dixin® 100 200 mg daily

4 R uwil id® 2 mg t i d orally

B V at um Compounds Th e compounds h ve n v r r c ved un-  
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small nd irregula be rptio f om th ga trolnte tinal tr i  
with resultant unpredict ble f lls in bl od p es re f thi  
con oral h xameth nium m oft n an tlef t ry

1 Basl p t t pl a

a Th pati t should be in th hospital unde lo s pe  
vi i

m Th infti l do should be m ll nd ould be inc m d  
gradually d pendi g pon the e tion of th pati t

m Th d gr of hyp t nsal h sh ld b only mod t t th  
fur t we k r and no attempt ould be m d t i r  
th pre re to normal until it ha be n d m trat d that  
the pati t an tol rate yst lic pre use f b i 160  
mm Hg witho t hypot nsal sympt ms

d The po t ral hypot i whi h is g t at t th h ight  
of the eff t of th d g should be onaid d not only  
a p t ilal da g to th pati nt b t i lo a th rap  
ti w pon to p along the hypot i ti of th d ug  
aft th pe k ff t ha worn off

- 2 Oral hexamethonium The initial dose is usually 125 mg of the ion and may be repeated at 6 to 8 hour intervals usually given 30 minutes before meals If no untoward effects or unusual hypotension occurs the dose can be increased to 250 mg t i d The dose can then be increased the average final dose usually approximates 2 to 3 Gm a day A trial of 2 or 3 weeks is usually required before the dose required to lower the blood pressure to a level approximating 180/100 can be determined

The patient may then be seen as an out patient and the dose gradually increased to that level which produces the desired fall of pressure Whether the desired level of pressure at the time of peak action is in the range of 150/160 systolic or is that which results in mild hypotensive symptoms on standing has not been determined Constipation must be avoided in patients receiving oral hexamethonium because it increases the absorption of the drug laxatives should be given to ensure a daily bowel movement

- 3 Parenteral hexamethonium The initial dose is usually 2.5 to 5 mg of the hexamethonium ion given subcutaneously If no untoward effect occurs the dose can be repeated in 12 hours On the second day 5 mg may be given twice at 12 hour intervals and the dose gradually increased On discharge from the hospital in 2 to 3 weeks the average patient receives approximately 75 mg of the hexamethonium ion twice daily In some patients it may be necessary to give the drug 3 times a day but the increments should always be made gradually and the patient observed several days before increasing the dose Particular caution should be exercised in older patients to avoid lowering the pressure too rapidly this is true also of those patients with evidence of atheromata in the cerebral or coronary arteries because acute hypotension may result in thrombosis of these vessels

In patients with cerebral or coronary arteriosclerosis Wood advocates the use of anticoagulants beginning a week prior to the administration of hexamethonium to lessen the danger of thrombosis

Following discharge from the hospital the patient should be seen at frequent intervals and the dose increased or adjusted so as to achieve the desired effect without undue faintness or side effects In some patients it may be necessary to have the patient lie down for an hour after each injection to prevent a postural hypotension which may produce severe symptoms during this period In many of these patients however tolerance gradually develops although marked hypotension may still occur on standing the patient may be able to sit or walk immediately after an injection Patients should be warned to avoid motionless standing for an hour or so after an injection shaving waiting in line for a bus and similar activities should be particularly condemned

- 4 Hexamethonium in acute hypertensive emergencies In acute hypertensive emergencies give hexamethonium intravenously at a rate of approximately 1 mg per minute to a total dose of 10 to 20 mg depending upon the response of the patient The most important of these is acute pulmonary

edema associated with a marked rise in blood pressure occurring in hypertensive patients with left ventricular failure. Dramatic improvement in the pulmonary edema under these circumstances may occur. Great caution must be used to give the drug slowly and to stop the administration when a moderate fall in pressure has been achieved. A further fall in blood pressure may occur for a time after the drug is stopped.

### 5 Side effects and hazard of hexamethonium

- a. Acute hypotensive falls in blood pressure are manifested by faintness, weakness and nausea and vomiting and the patient should be instructed to lie down immediately when these occur and place his feet higher than his head. Unless the hypotensive effect is too severe the symptoms pass off rapidly with this positional assistance. If the symptoms and the severe hypotension persist give a vaso-pressor drug such as N osynephrine® or Vasoxyl® subcutaneously or a slow central catheter infusion intravenously of 1 art emol 4 mg /liter (a t p 33). If the dose of hexamethonium is varying gradually increased the acute severe hypotensive falls are in most cases avoided.
- b. Acute or progressive renal failure due to decreased renal blood flow or filtration pressure may require discontinuation of the drug. If the dose is increased gradually this is usually obviated.
- c. Vascular thromboses are a hazard in older patients who suffer severe hypotensive falls but if the precautions outlined above are taken this complication is quite rare.
- d. A low sodium diet potentiates the action of hexamethonium and if an individual receiving fixed doses of the drug is given a low sodium diet hypotensive symptoms may occur. To obviate this it is usually desirable to place the patient on a 1-3 Gm sodium diet at the onset of therapy.
- e. Alcohol, hot liquids and vasodilator drug potentiates the action of hexamethonium and the possible side effects of danger should be kept in mind.
- f. Para sympathetic effects (due to para sympathetic blocking) Blurring of vision, constipation and dryness of the mouth can be corrected in part by the use of atropine orally in 7.5 to 15 mg doses.

### 5.1 Procedures

- A. Sympathectomy The therapeutic value of sympathetic has been highly controversial although most authorities agree it prolongs life when used in severely malignant hypertension good renal function.
- B. Adrenalctomy This radical procedure has been tried in patients with severe hypertension. The results have not been impressive though some patients with severe hypertension have considerable benefit.

### Low sodium Diet

A rigid low sodium diet containing 350 mg of sodium per day has been recommended. Popularized it is effective. Portion of case in which diet is rigorous and many



## 162 Hypertensive Cardiovascular Disease

difficult to continue the diet for the months and years required at present it would seem to be indicated only in the moderate forms of hypertension in which drug therapy is not to be used. The diet should also be used as an adjunct to the use of the hypotensive drugs. For details of the diet see p. 53.

### Psychotherapy

Considerable evidence is available to indicate that the hypertensive patient often has emotional conflicts particularly of the sort related to expressions of hostility and of dependence and independence. Emotional disturbances resulting from the existence of the hypertension itself must also be considered. Such emotional disturbances are undesirable because they aggravate the degree of existing hypertension and increase the loads on the heart and kidney. Attempts have been made to treat hypertensive patients with psychoanalytic methods. But the effect on the blood pressure has been poor even though symptoms may often be improved. Reversal of hypertension following psychotherapy has been extremely rare. Attention to the emotional needs of the patient is an important adjunct to other methods of treatment but should not be the sole method of treatment except in the mild benign forms of the disease in which drug or surgical therapy is not indicated.

### Other Methods of Treatment

- A Sedation Nervous tension is frequently found in the hypertensive patient and may aggravate his illness. In many cases sedation either used alone or as an adjunct to other forms of medical therapy will be of decided benefit. Phenobarbital is the drug most commonly used. Dosage 15-30 mg ( $\frac{1}{4}$ - $\frac{1}{2}$  gr) t.i.d. to q.i.d.
- B Drugs which have evoked little general enthusiasm despite occasional favorable results because of the unpredictable effects on the hypertension and the high incidence of unpleasant side effects include Dibenamine<sup>®</sup>, the dihydrogenated ergot preparations, Priscoline<sup>®</sup> and potassium thiocyanate and the long acting nitrite.

### Treatment of Complications

The cardiac, cerebral and renal complications of hypertension are discussed under congestive failure (see p. 182), angina pectoris and myocardial infarction (p. 183), cerebral hemorrhage and thrombosis (p. 349) and renal failure (p. 301).

### Headache

Much of the headache of hypertension is of an emotional type. Suggestion and explanation is often helpful. Hypotensive drugs are most effective in relieving severe headache associated with the malignant or pre-malignant phase of hypertension.

## CORONARY HEART DISEASE

Cor na = In uff i cy

Co onary in uff y s a dynam c co ept wh h i one rn d with th balance between the blood fl w in the m a y a i a d the d ma m f th my rdium f bl od Co ona m nauff c ency xlt wh n v th r quirement of th myoca diam for vygenated blood eds the fl w of blo d of th myoc rd um at any in ta t

Co onary insuff ency m y be c te and t m ent n whi h ca it is giv n th nam fa gina p t s t may b acute pro tracted and m ocated with myoca d al nfarct on o it may be sub ut mod tly p otracted a d witho t myo dual n is b t lincally m gn bl and thl ha b n all d oronary failure by Blumga ta d hu oon tes and is tho ght to repres t o l n f a l t ly m n r co na y t yw th uff t ollat ralc r ulat nt p v t my c rd l n c ois m should be t at d aim l ly t a mild infe ction

## ANGINAL SYNDROME

(Angi a Pectoris) (code No 401)

On m st d g n se angina p to by p a t v d gnost i te i t by x l s on Th diagno d p d p n p op t p station of car f l histo y and an a u ate evaluat on of th r d b l ty of th p tle t

Diagn s

Th d nal ymptom of th a ginal ynd m i pain ind d by ything that inc as s th w k of th h rt Pain i ually a b t rnal b t m y b p dual The o set l s dden b i not in stant us if cha t is that f p es m a eq s i g a sena ti n f a gnaf ant b t aho t d at n a ly la ting mo th n 15 20 m t s and p ec p tat d by ti n e item nt ld nd m vy m la

T m t f the A t Atta k

A Spe ll M Th nit II are the d gs employed

1 Gly ylt nitrat (nit ogly ine) is the d g of choic it cts in sbo t l 2 mi t Aa soon s att k b g s pla a o 3 mg tablet (1/200 g ) unde th to gu and allow it to d o l The d m y b incre sed to 0 4 0 6 mg (1/150 1/100 gr ) if n h f i bt ined from a small d Nit ogly rin m y b us d f ly wh eve an ti k oc so may be s d in rd t p e ent a t tack lt may ca head h

2 Amyl nit it 1 pearl rushed nd inhaled s t in about 10 se ds Thi d g usually c s s d is gr bl m

a ti one of flushi g of the fa ounding of th p ise and a metim dizzia s a d he dache Th r t on may b miniml d by inhaling th drug f om a distan o by rap dly pa ing th c h d p l b fore th no Th pati nt oon le rns h w to ary th am t of d gh wish to inhal

3 Lo ger-a ting nit ties and oth r drugs have no pla i th the py of the s te atta k

## 164 Anginal Syndrome

4 Alcohol 30-60 cc (1-2 oz) of whisky Brandy etc may be a helpful home remedy

- B General Measures. Rest is the most important therapy in an attack. The patient should cease any exertion and should stand still or sit or lie down as soon as he detects onset of pain and until the attack is over. This generally is the natural reaction of most patients but some try to work the attack off. Patients should be warned against this.

### Prevention of Further Attacks

#### A Specific Measures

##### 1 Drugs

###### a Nitrites

(1) Longer acting nitrites. Pentaerythritol tetranitrate (Peritrate®) seems to be the most effective of these. The average dose is 10 mg t.i.d. a.c.

(2) Glyceryl trinitrate (nitroglycerine) 0.3-0.6 mg (1/200-1/100 gr) under the tongue just before activity.

b Xanthines. These drugs may be of some benefit orally in large doses (see p. 204).

c Khellin (visammin). Its use has been recommended but the reported results are not convincing. Average doses are 40-240 mg orally. There is a high incidence of toxic reactions mainly nausea vomiting and dizziness.

2 Abdominal support. Obese patients with protuberant abdomens who have a gynaecological condition may have fewer attacks following the use of proper abdominal support. The mechanism is not clear. The Kerr-Lagen belt is designed for such purpose.

3 Surgical procedures. These have been employed only in patients with severe incapacitating angina pectoris in whom medical treatment has failed. The results are not good and the various surgical procedures that have been performed in the past are rarely used today.

4 Production of myxedema by means of thiouracil compounds or radio active iodine (Iodine 131) (see p. 372).

The object of this treatment is to produce a myxedematous state so as to reduce the work of the heart. Good results have been reported in about half of the cases of intractable angina but this method should not be used until prolonged rest and attention to the emotional needs of the patient have made it certain that one is not dealing with a transient reversible coronary insufficiency.

#### B General Measures

1 Avoid excesses. The patient must avoid all habits and activities that he knows will bring on an attack.

2 Treatment of coexisting disorders especially anemia which may lead to increased cardiac ischemia.

3 Rest. Most patients with a gynaecological condition do not require prolonged bed rest but rest and relaxation are beneficial. Adequate mental rest is also important.

4 Diet. Obese patients should be placed on a reducing diet and their weight brought to normal or slightly subnormal levels. Some authorities recommend a rigid low cholesterol low fat diet but the prevailing opinion is that a reducing diet is equally effective. The exact role of a special diet in

influencing the course of angina pectoris remains to be determined.

- 5 Tobacco is best avoided or used in moderation because tobacco produces tachycardia and elevation in blood pressure.

## ACUTE MYOCARDIAL INFARCTION (code No 430 516 7)

Myocardial infarction is due to necrosis of a portion of the cardiac muscle as a result of impairment of its blood supply. This impairment usually results from occlusion or thrombosis of a coronary artery but it may result from impaired blood flow as a result of shock or a vascular spasm from any cause. Myocardial infarction varies qualitatively from histologic necrosis to massive infarction. All intermediate degrees occur and result in varying clinical effects. The infarction may be essentially asymptomatic.

The onset of angina pectoris may be associated with coronary occlusion even though infarction does not occur (if the collateral blood flow is adequate). The prognosis is usually better than previously thought.

### Diagnosis

In typical cases the infarction is heralded by severe prolonged retrosternal pain similar in location and radiation to that of angina pectoris. It is often associated with shock, congestive failure and arrhythmias. Delayed manifestations include fever, leukocytosis and elevated sedimentation rate. ECG abnormalities are typical with characteristic changes in Q, T and ST segments which may be delayed. Variations from the typical pattern are not uncommon.

### Treatment

#### A. Immediate Treatment

- 1 Rest. Physical and mental rest is the most important position is essential during the first 2-3 weeks during which time rupture of the heart is most apt to occur. The patient should not be allowed to feed or care for himself during the first few days unless a physician is very mild with no shock or other complications. Spontaneous sweating is highly desirable. A bed table commodes are now considered a requisite so that the use of a bedpan (unless the patient can use the latter satisfactorily).

#### B. Relief of Pain

Morphine sulfate U.S.P. 20-15 mg (½-¼ gr) slowly I.V. (analgesic potentiation on lungs) if the pain is not relieved in 15 minutes repeat this dose. Although the initial relief of pain further intensification of morphine can be given up to 2-15 mg (½-¼ gr) as necessary for relief of pain. The subcutaneous route of injection is used until the effect is over then the patient is in the hospital. If the patient is in shock with vascular pain the venous use of morphine may be necessary because of poor absorption of the drug when administered orally. CAUTION: Do not give a second morphine if respirations are below 12.

- Demerol® and Dilaudid® are preferred to morphine by some clinicians in the treatment of infarction they are said to produce less nausea and vomiting
- Dihydromorphinone Hydrochloride U S P (Dilaudid®) 4 mg ( $\frac{1}{16}$  gr) I M or I V may be used instead of morphine
  - or c Meperidine Hydrochloride U S P Pethidine Hydrochloride B P (Demerol®) 50 100 mg I V or I M as needed
  - d Aminophylline U S N B P 0.5 Gm ( $\frac{1}{2}$  gr) I V very slowly (1-2 cc per minute) may be helpful if the pain is not relieved by opiates and/or oxygen (see below)
- 3 Oxygen Often useful and sometimes necessary for the relief of dyspnea cyanosis pulmonary edema shock and chest pain Give by BLB OEM or Bennett mask oral pharyngeal catheter nasal metal inhaler or oxygen tent (see p 147)
  - 4 Reassurance From the onset attempt to allay patient's apprehension and anxiety
  - 5 Shock A frequent and serious complication with an estimated mortality of 80% particularly in those in whom shock is delayed and appears after the pain has subsided
    - a Vasopressor drugs Present evidence suggests that vasopressor drugs (sympathetic amines) may elevate the blood pressure and decrease mortality in myocardial infarction associated with shock Shock may be treated early to achieve the best results For details of the use of vasopressor drugs see p 33
    - Digitalis A hypotonic myocardium often accompanies acute myocardial infarction and shock may be associated with an increased venous pressure Contrary to previous opinion some investigators are impressed with the value of digitalization in the shock of acute myocardial infarction Digitalization can be accomplished as in congestive heart failure The increased cardiac output results in increased coronary flow and the pressure may rise
    - c Treatment of cardiac arrhythmias Shock may be the result of undetected ventricular tachycardia or other arrhythmia and prompt treatment of this complication (see below under cardiac arrhythmia) may be life saving
    - d Venous and arterial transfusions These have not been very effective but should be kept in mind as adjuncts
  - 6 Anticoagulant therapy This is a controversial matter despite the official recommendation of the American Heart Association that anticoagulants decrease the mortality and the incidence of thromboembolic phenomena in acute myocardial infarction Other investigators have challenged this especially in the milder cases (rapid relief of pain minimal signs of myocardial necrosis absence of shock or cardiac failure) In severe cases of myocardial infarction anticoagulants are generally recommended For technique see p 216
  - 7 Sedation Adequate sleep is as vital in patients with myocardial infarction as it is with those suffering from cardiac failure Whatever drugs necessary should be used to provide

sufficient analgesia and morphine derivatives should not be withheld in the first few days if they are indicated.

- D Follow-up** Careful clinical observation is mandatory to determine the patient's progress. One should be alert for evidence of extension of the infarction, new infarction, the appearance of complications, or symptoms requiring treatment.

**E Treatment of Complications**

- Cardiac Failure** If cardiac failure develops, treat as for failure from any cause. Oxygen, low sodium intake, mercurial diuretics, and cautious digitalization are the essentials. The patient should be digitalized in such a manner as to avoid toxic reactions if possible. Rapid digitalization is best avoided unless the failure is urgent. If the cardiac failure is mild and manifested solely by pulmonary rales and increased dyspnea, restriction of sodium and the administration of mercurial diuretics may be sufficient. Digitalis is avoided by some authorities because of the hazard of ventricular arrhythmias. If its well-controlled administration should not be deferred if cardiac failure demands it.
- Arrhythmias**
  - Ventricular premature beats** These are common and indicate increased irritability of the damaged myocardium and may presage ventricular tachycardia. Quinidine sulfate is the drug of choice (see p. 200). An alternative to quinidine is procainamide (see p. 205).
  - Ventricular tachycardia** is an emergency (see p. 178).
  - Atrial fibrillation** is usually transient. If it persists, if the patient tolerates it poorly, or if congestive heart failure occurs, the patient should be digitalized with care (see p. 197).
- Adams-Stokes Attacks** with bradycardia or complete heart block (see p. 181).
- Thromboembolic phenomena** are common during the course of myocardial infarction. If anticoagulants have not been given, they should be promptly administered if thromboembolic phenomena occur (see p. 218).
- Extinction of the Infarction** When the severe chest pain has subsided, tension of the myocardial infarction should be suspended and the patient mobilized in the first 24 hours, and in other clinical features. The same methods of treatment apply to the transmural infarction, but if the requirement of rest is required.

**A. Activity Status in Convalescence**

The minimum period of bed rest should be at least 3 weeks. If the infarction has been very severe, this should be increased to approximately 6 weeks. The program for most patients is 1 month of complete rest, 1 month of slowly increasing activity, and a third month of restricted activity prior to returning to work. The amount of rest should be individualized according to the severity of the myocardial infarction and the response of the patient.

The patient should not be permitted to walk freely about the room for about 7-10 days after he is first allowed out of bed. Gradual resumption of activity is most important. Rest on the same floor with gradually increasing periods.

always slowly and without producing chest pain dyspnea undue tachycardia or fatigue When he is first permitted out of doors usually not until 2 months after the infarction he should avoid hills and stairs for another month

## CHRONIC RHEUMATIC HEART DISEASE

Rheumatic heart disease is one phase in the rheumatic fever cycle The stage of asymptomatic valvular heart disease without cardiac failure is the latent period between the subsidence of acute rheumatic fever and the terminal phase of cardiac failure The physician endeavors to prolong this latent phase as much as possible

### Management of Asymptomatic Valvular Heart Disease

#### A Prophylaxis

- 1 Prevention of recurrences of acute rheumatic fever
  - a Avoid exposure to streptococcal infections
  - b Continuous antibiotic prophylaxis in selected cases
  - c Prompt adequate treatment of hemolytic streptococcus infections
- 2 Prophylactic advice in regard to dental extraction urologic procedures surgical procedures etc to prevent bacteremia and possible subacute bacterial endocarditis

#### B General Measures

- 1 Proper vocational guidance to anticipate a later period when exercise tolerance may be significantly limited
- 2 Early recognition of disturbances of thyroid function anemia paroxysmal arrhythmia etc so as to provide proper therapy
- 3 Maintenance of general health at as high a level as possible with good habits adequate diet constant level of activity and adequate sleep
- 4 Avoidance of obesity and excessive physical exertion

## MITRAL VALVULAR DISEASE

This is the most common of valvular lesions It takes from 3 to 5 years for mitral stenosis to develop mitral insufficiency may occur alone or more commonly in combination with mitral stenosis

### MITRAL STENOSIS (code No 498)

In view of the excellent results obtained following mitral valvulotomy the signs of mitral stenosis should be clearly appreciated

#### Diagnosis

- A Signs of Uncomplicated Mitral Stenosis The most important of these are (1) a mid diastolic long murmur always associated with presystolic accentuation if the rr is sinus rhythm and usually associated with a thrill (2) a systolic murmur which if present is usually grade II or less and is not pansystolic (3) a snapping 1st sound and an opening snap

If pulmonary hypertension is present the signs of this and associated right ventricular hypertrophy may be demonstrated.

- B Exclusion of Mitral Insufficiency** Mitral incompetence must be excluded if possible. The mitral valve is operable only if the patient's condition is due to a mechanical obstruction of the mitral valve. If there is no aortic or systolic murmur in the precordium the signs of mitral stenosis, mitral incompetence, are exceedingly unlikely. If there is a loud pansystolic murmur at the apex in association with an aortic aortic often early 3rd heart sound, a soft 1st sound, and no opening snap, the diagnosis of predominant mitral incompetence is likely even if a short mid-diastolic murmur can be heard at the apex. Left ventricular hypertrophy on the ECG should make one very cautious in recommending surgery for mitral stenosis because of the likelihood of significant mitral incompetence. If there is a loud aortic systolic murmur at the apex the diagnosis must rest on a consideration of the total findings.

### Surgical Treatment

The course of mitral stenosis is highly variable and a view of the mortality of mitral valvulotomy (35%) surgery is not advised in mild cases with slight exertional dyspnea and fatigue only. Indications for surgery include the following:

- 1 Uncontrollable pulmonary edema
- 2 Disabling dyspnea and occasional pulmonary edema
- 3 Evidence of active pulmonary hypertension with right ventricular hypertrophy and early congestive failure
- 4 Systemic and pulmonary emboli
- 5 Increased pulmonary artery pressure in association with marked apnoea and increased P. These patients are prone to develop right heart failure and emboli
- 6 Right heart failure with atrial fibrillation and supraventricular tachycardia secondary to marked mitral stenosis. The diagnosis of mitral stenosis is difficult without these circumstances and the surgical mortality high.

### **AORTIC STENOSIS (code No 499)**

Within the past 2 years operative relief of aortic aortic stenosis has been successfully achieved.

#### Diagnosis

- A History and Findings** The criteria for clinical diagnosis of aortic stenosis are (1) loud rough aortic systolic murmur and thrill ending before the 1st sound and transmitted to the neck and apex (2) a heaving apical impulse typical of left ventricular hypertrophy (3) a small sustained and anacrotic pulse and a weak A<sub>2</sub>.
- B Other Symptoms and Signs** The patient may be asymptomatic or associated with dyspnea progressing to right heart failure, syncope, and/or angina pectoris.



Surgical Treatment

The indications for surgical correction of aortic stenosis are progressive left ventricular failure attacks of syncope due to cerebral ischemia and angina pectoris when thought to be due to the decreased cardiac output of aortic stenosis and not to associated coronary disease. In the presence of both mitral and aortic stenosis surgical correction of both valves can be performed at the same operation.

**BACTERIAL ENDOCARDITIS**

(Subacute code No 450 100 (Acute code No 450 100)

These are infections occurring on the interior surfaces of the heart (most frequently the valves) or great vessels associated with showers of large and small mycotic emboli. The most common infecting organisms are *Streptococcus viridans* and *Streptococcus faecalis* which cause the classical subacute bacterial endocarditis. Bacteremias which may accompany primary infections such as pneumonia may cause acute bacterial endocarditis due to pneumococci staphylococci beta hemolytic streptococci *Haemophilus influenzae* and gonococci.

Diagnosis

Symptoms are usually those of septicemia the signs include fever pallor petechial hemorrhages splenomegaly clubbing of fingers and evidence of valvular or congenital heart disease. Do not make a diagnosis of subacute bacterial endocarditis in the absence of valvular or congenital heart disease or without repeated positive blood cultures showing the same organisms. Do not exclude the diagnosis of bacterial endocarditis without repeated negative blood cultures or marrow cultures. Occasional typical clinical cases will be found in which there are repeatedly negative blood cultures. Under such conditions therapy must be started empirically to prevent serious damage to heart valves.

Treatment

A. Specific Measures The most important consideration in the treatment of bacterial endocarditis is a bactericidal concentration of one or more antibiotics in contact with the infecting organisms which are often localized in avascular relatively inaccessible foci. Penicillin because of its high degree of bactericidal activity against the great majority of bacteria which produce bacterial endocarditis and because of its low incidence of side reactions is by far the most useful drug. Synergistic combinations of penicillin with other antibiotics have often proved valuable. Few cases have been cured by bacteriostatic drugs such as chlortetracyclins (Aureomycin®) oxytetracycline (Terramycin®) chloramphenicol (Chloromycetin®) and erythromycin (Erythrocin®) used alone. Positive blood cultures are invaluable to confirm the diagnosis and to guide treatment and should be combined with tests of sensitivity of the infecting organism to various antibiotics or combinations of antibiotics. Hence one or more blood cultures should be obtained daily for 3 to 5 days before instituting

treatment ext pt in d sp rarely ill patient or patients with c to bacterial endocarditis To v id fa ther be t damag treatment h uld n t be further delayed

i Penicillin This d ug an t b g en parenterally in bac t rial endocarditis in o d r t gain eff ctive levels The dos of penic illin used depends on the sensit vity of the o rganism nd this is d termined by doing in vitro sensit vity t sts About 90% of t ins of Streptococ us virid ns f om c se of ub cute ba t rial endoc d st s have b n found t be inhibited in vitro by 0 1 unit of penicillin per c or le s How v r some re qu t f t nt r quiring up to 10 units or more

A minimum s ram on e t ation m ny tim s gr t than the appa ent in vitro sensit vity f the organism must b produced to insur a ba t ricidal con centration n th g tation In pati nt in whom posit ve blood cultur s are not btain d or whe e a sensit vity t sts a not av labl 5 to 10 million units of peni illin houl d be giv n daily The e are thr alternativ m thod of admini tr ti n

- a Peni illin proc line Fo organism s sensit v t less than 0 1 U p r ml of penicillin giv 500 000 to 1 000 000 nits of penicillin p ocal e t M twice daily
- b Int mittent administ ion Fo s gant m ensitive to 0 1 U pe ml of penicillin o mo s intermitt nt intra mus ula inye tions of aqu u p ni lln ol tion ev ry 3 t 6 h urs

or Continuous pe nt ral administ tio If th total daily dose is approximately 3 milli n s mo unit of peni illin day dministration i usually be t compli h d by a continuous int amuscular drip ( occ ionally i t s n u d ip ) Th antibi t c n be d s s lved n 1000 2000 of phy l l gical s lin s l t on o glucos lu ti n

#### APPROXIMATE DOSAGE SCHEDULES

Pe i illin inhib tion (Ba tericidal at 72 Hr ) Unit p r ml	Total m m d n Do ag pe 24 Hr (Milli of Unit )
0 1	1 2 (p illin p ocal n )
0 1 0 4	3 4 ( q uous )
0 5 0 9	4 5 ( q )
1 0 1 0	6 20 ( q uous )
> 5 0	20 500 ( q uous )

Wh n bact mia and f p r i t th dos ge h uld be doubl d nd do bl d until f o abl pon oc Alternat v ly vn g i t i tre m i w th 2 or m biot m y b d Wh h gh once t ations of re quired Proben id N N R (B m d") 0 5 (71 g ) e y 6 ho r m y b e u d to inhibit nal c i n

2 St ptomycin Int nite i i M in j ti is th

choice and gives as good levels as those obtained by I V injections. Large doses are advised. 0.5-1.0 Gm dissolved in 4 cc (1 dr.) distilled water + 1 cc 2% procaine I M every 6 hours should be given. Observe for toxicity.

- 3 Combined penicillin and streptomycin. Preliminary evidence suggests that penicillin (5 million units/day) + streptomycin (2 Gm /day) may be the optimal treatment for infections due to *Streptococcus faecalis*.
- 4 Chlorotetracycline (Aureomycin®), oxytetracycline (Terramycin®), chloramphenicol (Chloromycetin®) and erythromycin (Erythrocin®). While these drugs may suppress the progress of subacute bacterial endocarditis, their use is frequently followed by relapse. Wherever possible, drugs exhibiting more pronounced bactericidal activity, i.e. penicillin and streptomycin, should be the first choice in treatment. The exact dosages and effectiveness of therapy have not been established. Nausea and vomiting result frequently from the oral administration of Aureomycin® and may interfere with treatment. In such cases the drug must be given I V in doses of 50-100 mg. or more every 6 hours.

Although *Streptococcus faecalis* is generally inhibited by Aureomycin® and Terramycin®, treatment with these drugs of endocarditis due to this organism is generally ineffective.

- 5 Other drugs. Neomycin, bacitracin and polymyxin may be used alone or in combination with other drugs where the organism is insensitive to less toxic antibiotics (see p. 314).
- 6 Combined therapy. In infections due to highly resistant organisms, synergistic pairs of antibiotics — determined by tests of bactericidal activity in the laboratory — may be used (see p. 498). Combined therapy should never be attempted without adequate laboratory control.
- 7 Duration of treatment. The suggested duration of therapy by various authorities is 2-8 weeks. Most patients should be treated for 4 weeks after sterilization of the blood stream. After therapy has been discontinued the patient should be carefully observed for recurrence by taking repeated blood cultures.
- 8 Recurrences. Most recurrences are observed within a week or two of the end of therapy. Occasional cases relapse months later. The diagnosis of recurrence must not be made on the return of fever and embolic phenomena alone; these may occur for up to 6-8 weeks after therapy has ceased. Positive blood cultures are essential for the diagnosis of recurrence. Before re-treating again determine the sensitivity of the organism and then give treatment with higher dosages for a longer period of time or use a different antibiotic. About 70-75% cures are now being reported.
- 9 Anticoagulants. It is generally agreed that the use of heparin or bishydroxycoumarin (Dicumareol®) in the treatment of subacute bacterial endocarditis is unnecessary and may be dangerous.

B General Measures. General supportive measures as for any severe infection with fever should be given.

C Complications and Treatment

- 1 Infarction. Caused by emboli breaking off from infected

area. The infarctions usually occur in organs in the systemic circulation but if the endocardial lesion is on the right side of the heart the embolus may be to the pulmonary circulation. Treatment is symptomatic.

- 2 Cardiac failure (uncommon). Active myocarditis or scarring of the heart valves may precipitate congestive failure. When giving large quantities of penicillin as sodium salt one may give significant amounts of sodium ion. Therefore when treating a case of subacute bacterial endocarditis with congestive failure or possible failure use calcium or potassium penicillin. (See congestive failure p. 182)
- 3 Anemia. The anemia if severe should be treated by whole blood transfusions (see p. 147)

### Prophylaxis

A high percentage of cases of endocarditis arise after dental procedures or surgery of the oropharynx and genitourinary tracts. Therefore all patients with valvular or congenital heart disease who are to have any of these procedures should be given penicillin prophylactically. A satisfactory schedule is as follows: Penicillin 1,000,000 units daily for 2 days before procedure on the day of the procedure and 2 days after the procedure.

## CARDIAC ARRHYTHMIAS

Cardiac arrhythmias are commonly found in every physician's practice and a thorough knowledge of their diagnosis and management is essential. Clinical manifestations vary from trivial palpitations to a clinical state of the utmost urgency as when ventricular tachycardia complicates acute myocardial infarction.

### Relation of symptoms

The symptoms produced by an arrhythmia depend upon the underlying state of the heart, the ventricular rate, and the duration of the arrhythmia. Even a normal heart may fail if the ventricular rate is rapid enough or lasts long enough. Tachycardia which may be well tolerated by one individual may produce severe pulmonary edema (e.g., in a patient with tight mitral stenosis). The physician must decide when to ignore, when to treat conservatively and when to attempt maximum efforts to correct the arrhythmia.

## DISTURBANCES OF AURICULAR ORIGIN

### AURICULAR PAROXYSMAL TACHYCARDIA (code No. 422)

In this discussion of arrhythmias it is thought that an ectopic focus within the auricle takes over as the pacemaker of the heart and discharges impulses at rates varying from 120-320 per minute between 170 and 210 per minute. The rate is usually irregular and is not affected by respiration, movement or emotion (unless this abolishes the attack). Auricular is usually a benign condition unless it complicates severe

disease. At least half of the cases occur in individuals without organic heart disease. Death during an attack is rare, but cerebral ischemia or cardiac failure may occur when the more rapid rates last over a period of days. The attacks are often produced by emotional tension, are common in young individuals, and at times are related to accelerated conduction through the A-V node, formerly known as the Wolff-Parkinson-White syndrome. Recurrent attacks are frequent, so that the problem of preventing attacks is as important as the treatment of the individual attack.

#### Treatment of the Atrial Attack

In the absence of heart disease one should remember that serious effects are rare. Most attacks tend to subside spontaneously, and the physician should not use remedies that are more dangerous than the disease. Particular efforts should be made to stop the attack quickly if it persists for several days, if cardiac failure, syncope, or anginal pain develops, or if there is underlying cardiac disease.

- A Mechanical Measures. A variety of methods may interrupt attacks, and the patient may learn to do these himself. These include the Valsalva maneuver (holding breath and contracting chest and abdominal muscles), stretching of the arms and body, lowering the head between his knees, and holding the breath.
- B Vagal Stimulation
  - 1 Carotid sinus pressure. With the patient relaxed in the semi-recumbent position, firm but gentle pressure and massage should be used, first over one carotid sinus for 10 to 20 seconds and then over the other. Pressure should not be exerted on both carotid sinuses at the same time. Continuous auscultation of the heart should be carried out, so that pressure is stopped as soon as the attack ceases. Carotid sinus pressure will interrupt about half of the attacks, especially if the patient has been digitalized.
  - 2 Bilateral eyeball pressure has been recommended, but it is rarely as effective as carotid sinus pressure and carries the risk of producing a detached retina.
  - 3 Induced vomiting (except in cases of syncope, anginal pain, or severe cardiac disease).
- C Drug Therapy. If mechanical measures fail and the attack continues (particularly if the symptoms noted above are present), drugs should be employed. There is no unanimity of opinion about the most effective drugs, but the following are satisfactory:
  - 1 Quinidine sulfate U.S.P. B.P. (see p. 200).
  - 2 Neostigmine U.S.P. (Prostigmin®) 1 mg subcutaneously.
  - 3 Digitalis orally, or, if no digitalis has been given in the preceding 2 weeks, intravenously.
  - 4 Procaine amide (Pronestyl®) (see p. 205). Continuous electrocardiograms or continuous monitoring of the heart rate and blood pressure is essential.
  - 5 Methacholine Chloride U.S.P. B.P. (Mechoyl® chloride) 10 mg subcut. is often effective but produces very unpleasant side effects and is usually contraindicated.
  - 6 Syrup of Ipecac U.S.P. 4 to 8 cc. may be used to induce vomiting. It may be repeated if unsuccessful.

Prevention of Attacks

A Attempt to find and remove the susceptible emotionally stressed and undue fatigue or excessive alcohol or tobacco

B Drugs

- 1 Quinidine sulfate U.S.P. B.P. 0.2 to 0.6 Gm (3 to 9 gr) 4 times a day may prevent further attacks if they occur frequently and are troublesome. Begin with small doses of quinidine and as if the attacks are not prevented and toxic effects do not occur.
- 2 Should quinidine fail or if it is not tolerated full digitalization followed by digitalis maintenance doses may prevent the frequency of attacks (see p. 187).
- 3 Maintenance dose of procainamide 250 to 500 mg tid may be tried if the above two methods are unsuccessful.

**NODAL PAROXYSMAL TACHYCARDIA (code No. 422)**

This syndrome is usually a tachycardia except that the ectopic focus is in the A-V nodal tissue. At times the electrocardiographic or clinical distinction is not between nodal and nodal paroxysmal tachycardia as possible, which is the former. Paroxysmal tachycardia is seen. The attack is usually along the same line as for nodal tachycardia (see p. 173).

**AURICULAR FLUTTER**

(Paroxysmal code No. 423) (Chronic code No. 424)

This arrhythmia is due to impulses which arise from an irritable focus of a nodal nature at rates of 250 to 350 per minute. The auricular rate is usually one half of the atrial rate (2 to 1 conduction) but the ventricular rate may be less (3 to 1 or 4 to 1 conduction) or rarely may be 1 to 1 conduction with a very rapid ventricular rate. The ventricular rate is usually regular but if the effect is significant A-V block the ventricular rate may be irregular and may stimulate a nodal fibrillation. Atrial fibrillation usually results from a frequent premature contraction and it may occur irregularly in the absence of heart disease. It may be produced by quinidine and is the same as the treatment of nodal fibrillation.

Treatment

- A Treatment of Paroxysmal Flutter. Similar to treatment of paroxysmal tachycardia except that digitalis and quinidine are the drugs of choice. The arrhythmia tends to be more established and more often nodal in origin. Prophylaxis for the attack is directed out similarly to that of nodal tachycardia (see p. 173).

B Treatment of Chronic Auricular Flutter

- 1 Digitalis is the drug of choice if there is an A-V block and prevents a 2 to 1 or 1 to 1 conduction in about half of the cases of nodal fibrillation. In arrhythmia sustained from digitalization if a nodal fibrillation is maintained after it has been produced by digitalis quinidine

be added to convert to sinus rhythm. Digitalis may be given by any one of the usual methods (see p. 197). Oral medication is usually sufficient although the intravenous route may be used if the situation is critical. Digitalis must often be given in larger doses than are usually required for cardiac failure. When a fixed 4 to 1 conduction is produced by digitalis a slightly increased dose may convert the flutter to auricular fibrillation or sinus rhythm.

- 2 Quinidine sulfate. This drug should not as a rule be used to treat auricular flutter unless the patient is fully digitalized with a slow ventricular rate because of the danger of producing a 1 to 1 conduction. If digitalis results in only a 4 to 1 conduction or produces auricular fibrillation which does not spontaneously convert to sinus rhythm quinidine may be given (see auricular fibrillation p. 176).

### AURICULAR FIBRILLATION

(Paroxysmal code No 425) (Chronic code No 426)

A common arrhythmia due to ectopic impulses arising in the auricle at very rapid rates (400-500) they often follow variable auricular pathways. The ventricular rate is always irregular most commonly varying between 110 and 160 but may be slower or faster depending upon the degree of A-V block. The chronic form is usually but not invariably associated with organic heart disease especially rheumatic mitral valve disease coronary and hypertensive heart disease and thyrotoxicosis. The paroxysmal type may occur without apparent reason in normal individuals in apparently normal hearts during acute infectious diseases following surgical operations especially of the lungs and particularly in thyrotoxicosis.

#### Treatment

#### A. Treatment of Paroxysmal Auricular Fibrillation

##### 1. Specific treatment

- a Digitalis is the drug of choice in paroxysmal auricular fibrillation especially when this arrhythmia occurs in individuals with organic heart disease (particularly mitral stenosis) with rapid ventricular rates or when the symptoms or signs of cardiac failure have appeared. If there is doubt as to whether one should use quinidine or digitalis first digitalis should be given this is because it controls the ventricular rate by producing an A-V block which is the immediate objective of treatment in such a case. The objective of treatment with quinidine is to abolish the auricular ectopic rhythm and it is quite safe to wait until the ventricular rate is brought under control with digitalis. Give full digitalizing doses (see p. 197) with the objective of slowing the ventricular rate to 70 to 80 per minute and avoiding toxic manifestations. In paroxysmal fibrillation there is no clear evidence that the use of digitalis will result in established fibrillation.
- b In those cases where an attack of auricular fibrillation persists in an otherwise normal heart with a ventricular

rate under 100 and with no other symptoms or signs of cardiac failure quinidine sulfate may be used at once to convert the rhythm to sinus rhythm.

If the ventricular rate becomes very rapid or if symptoms of dyspnea, anginal pain or severe palpitations are produced conversion with quinidine should be temporarily suspended and digitalis given.

- 2 Prophylaxis of paroxysmal fibrillation. The principles and procedure are the same as for auricular paroxysmal tachycardia (see p. 173).

## 8 Treatment of Chronic Auricular Fibrillation

### 1 Drugs

- a Digitalis. The high digitalization is the first step (see p. 127). The patient is then usually placed on maintenance digitalis indefinitely. The object of digitalization is to slow the ventricular rate and to improve myocardial efficiency.
- b Quinidine sulfate. Quinidine is used to abolish the tachycardia once the ventricular rate is controlled with digitalis. It is potentially hazardous and should be used only in carefully selected cases by physician thoroughly familiar with the drug and by a method which ensures close medical supervision (preferably in the hospital) while conversion to sinus rhythm is being attempted.

CAUTION: See p. 200 for danger of quinidine.

- 2 Conversion of chronic auricular fibrillation. Current opinion varies about the following indications for conversion of auricular fibrillation to a regular sinus rhythm. Each case must be individually considered. In general conversion is attempted whenever it is thought that the patient will be better off with sinus rhythm than with auricular fibrillation.
  - a Auricular fibrillation persisting after thyrotoxicosis has been treated surgically or by other means.
  - b Auricular fibrillation of a few weeks' duration in an individual with no or only slight cardiac disease.
  - c Auricular fibrillation associated with frequent embolic phenomena.
  - d Refractory cardiac failure induced by the auricular fibrillation.
  - e Severe palpitation and inability to decrease the ventricular rate with digitalis therapy be obvious only on exertion.
  - f Auricular fibrillation appearing for the first time postoperatively in patients with a technically successful mitral valvulotomy.

## DISTURBANCES OF VENTRICULAR ORIGIN

### VENTRICULAR PREMATURE BEATS (code No. 441)

Arrhythmias in which ectopic impulses arise from a point in the ventricle to cause a premature beat. It is one of the most common arrhythmias and often occurs in individuals without heart



TreatmentA Emergency Measures

- 1 Position The patient should be elevated to the semi Fowler bed position (see p 3) or put in a chair this decreases the venous return to the heart
- 2 Morphine sulfate 15 to 30 mg ( $\frac{1}{4}$  to  $\frac{1}{2}$  gr) I V or I M relieves anxiety and depresses pulmonary reflexes and induces sleep The attendant lessening of the forceful respiration decreases the negative intrathoracic pressure and the venous return to the heart
- 3 Oxygen when available should be administered in high concentrations This is best achieved by mask or in the case of children by hood or tent Mod rate concentrations (40 to 60%) can be achieved by oxygen tent or nasal catheter Oxygen relieves hypoxia and dyspnea and decreases pulmonary capillary permeability (see p 145)
- 4 Reduction of blood volume
  - a Tourniquets Soft rubber or blood pressure cuffs applied with sufficient pressure to obstruct venous but not arterial flow and rotated every 15 minutes will effectively reduce the venous return to the heart The tourniquets should be removed gradually as the attack subsides Approximately 700 cc of blood may be trapped in the extremities by this method
  - b Venesection (300 to 700 cc) This is the most direct way of reducing the venous return to the heart and may strikingly increase the cardiac output and decrease the right auricular and peripheral venous pressure in low output cardiac failure This procedure should not be done if anemia is present
- 5 Rapid digitalization is of great value (see p 197) Great care should be exercised in giving digitalis intravenously to a previously digitalized patient
- 6 Aminophyllin 250 to 500 mg slowly I V has been advocated Oral aminophylline is relatively ineffective I M aminophylline is often painful Rectal aminophylline suppositories 0.5 to 0.5 Gm ( $\frac{3}{4}$  to  $1\frac{1}{2}$  gr) may be helpful
- 7 Hexamethonium In the acute pulmonary edema of hypertensive heart disease and in the presence of severe hypertension a slow intravenous infusion of hexamethonium 1 mg per minute to a dose of about 5 to 10 mg may be very helpful The infusion should be stopped when the systolic pressure falls to 170 so as not to overstep the mark and produce hypotension

**REFRACTORY CARDIAC FAILURE**

The refractory state is said to be present when the patient fails to obtain clinical improvement after the usual therapeutic measures outlined above When this occurs the following procedure is advised

- 1 Re-evaluate the total situation Has the bed rest been adequate? Is the patient getting more sodium than ordered? Has he been receiving his therapy fully? A review of the patient's activities diet and medications is essential



## 188 Acute Fibrinous Pericarditis

per day for 3 to 4 days and repeat after a rest interval of 3 to 4 days

- 2 Potassium salts may be added if a deficit exists (see below) If tetany is present calcium salts must be given concurrently (see p 302)

### Low Sodium Syndrome

A Diagnosis The onset of weakness oliguria diaphoresis and azotemia heralds the low salt syndrome Hot weather fever and vomiting are additional predisposing factors Low serum sodium may be present without alkalosis or acidosis or it may be complicated by dehydration and acidosis It may follow severe sodium restriction accompanied by mercurial diuresis

### B Treatment

- 1 Mild cases Increase sodium intake  
2 Severe cases Treat with I V hypertonic saline (see p 31)

### Hypokalemia

A This may result from excessive potassium excretion from mercurial diuresis or the use of resins in patients receiving a low sodium diet Hypokalemia may induce digitalis intoxication and is manifested by muscular weakness particularly of respiration

B Treatment Potassium chloride 4 to 8 Gm (1 to 2 dr ) daily by mouth provided there is no renal failure CAUTION Parenteral potassium salts should not be given in the presence of acidosis or renal failure

## PERICARDITIS

### ACUTE FIBRINOUS PERICARDITIS (code No 420 )

Acute fibrinous pericarditis may be caused by or associated with many diseases The most common are those due to rheumatic fever uremia tuberculosis viruses and malignant disease Acute fibrinous pericarditis generally produces little functional impairment for there is no mechanical interference with cardiac function The most distressing symptom is pain and this may be entirely absent It varies from local discomfort to very intense pain which is usually substernal or precordial and may be confused with angina or infarction

### Treatment

Treat the underlying condition and provide analgesics as necessary for relief of pain Salt tablets and/or ACTH or cortisone are useful in rheumatic pericarditis (see p 518)

# PERICARDITIS WITH EFFUSION (code No 420 100 8)

The diagnosis of pericarditis with effusion is important for the fluid accumulation may cause cardiac tamponade. This does not respond well to the usual measures of the treatment of a fluid re (i.e. digitalis, low salt etc.) but removal of the pericardial fluid may be lifesaving. Thus it infrequently requires drainage. The most varieties of pericardial effusion with the exception of tuberculous pericarditis. Both the rapidity with which the fluid accumulates and the amount of fluid is important in determining the functional impairment.

## Differential

Symptoms and signs include peripheral dyspnoea, cyanosis, distended neck veins, tachycardia, pulsus paradoxus in the absence of cardiac dullness, a friction rub and diminished heart sounds. Pericardial friction rub may be present. The heart may be enlarged, bottle-shaped heart shadow. The ECG shows low voltage of the QRS complex and T wave flattening. Diagnostic aids include chest x-ray, echocardiography confirm the diagnosis by pericardiocentesis.

## Treatment

**A. Emergency Treatment (Pericardiocentesis)** The indication for pericardiocentesis is the symptomatic signs of cardiac tamponade. As the pericardial fluid increases in amount and pressure rises when it is rapidly reabsorbed, the pressure may rise considerably and the cardiac output may progressively fall. When this occurs the patient becomes weak and dyspnoea, the pulse pressure becomes very narrow and the pulse rapid and thready. The patient goes into shock. Under these circumstances the removal of the pericardial fluid may be lifesaving. The fluid should be removed slowly to avoid cardiac dilatation or serious bleeding.

**1 Sites for puncture** Avoid puncture of the ventricular muscle.

Left 5th or 6th intercostal space 1 cm within the area of cardiac dullness. 1-2 cm inside the left heart border as localised by x-ray (usually 7-8 cm outside of left sternal line). The needle is pushed slowly and slightly upward. If friction is present on hold of fluid within 3-5 mm (1-2 cm). 7-8 cm.

**B. Epigastric area** A horizontal incision 1 cm above the sternal margin. Insert the needle downward at a depth of about 30 cm and point it towards the midline. The pericardium is reached at about 3-4 cm.

**C. Pericardiocentesis** To be performed only when the above approaches remain unsuccessful. Reclined if one suspects a pleural effusion. On the left 7th or 8th intercostal space in the mid-pulmonary line. The left arm is elevated to rotate the scapula out of the way. The needle is directed inward and medially.

## **2 Equipment**

No. 16-18 gauge needle with sharp bevel and fitting stylet.

## 190 Purulent Pericarditis

- b No 26 or 27 needle to infiltrate the skin with procaine
- c 20 III cc syringe to remove fluid Syringe should be connected to needle by a 4 inch piece of rubber tubing to prevent excessive movement of the needle

### 3 Technic

- a Clean and sterilize skin over area to be punctured
- b Drape surrounding area with sterile towels
- c Infiltrate skin with 1-2% procaine solution
- d Insert needle (detached from syringe and without a stylet) slowly into skin following directions according to site selected (see above)
- e Withdrawal of fluid When the fluid is encountered it must be withdrawn very slowly sudden withdrawal of the fluid may result in acute cardiac dilatation failure or death Some consider it advisable to replace half the amount of fluid withdrawn with air both to prevent excessive dilatation and to give better visualization of the process by x ray With the needle in place remove III cc portions after the withdrawal of each portion inject 10 cc of air
- f After the needle is removed a simple bandage over the needle puncture is adequate

### B Specific Measures

- 1 Tuberculous pericarditis (code No 420 133) The current treatment is to treat the systemic tuberculous infection with bed rest attention to nutrition and other general factors and intensive anti tuberculous drug therapy If the fever and signs of pericardial effusion do not rapidly subside and are still obvious in a month surgical decortication of the pericardium should be considered in order to prevent chronic constrictive pericarditis Judgement is required to determine when the disease is progressing despite medical treatment and when signs of constriction are appearing
- 2 Rheumatic pericarditis with effusion (code No 420 196 8) Treat as for rheumatic fever The salicylates may help in causing fluid resorption Paracentesis is usually unnecessary but should be performed if tamponade occurs
- 3 Hydropericardium due to heart failure (code No 420 522 8) Treatment of the congestive failure is usually sufficient
- 4 Hemopericardium due to rupture of adjacent structure (code No 420 532) Usually post traumatic If fluid accumulation is excessive remove fluid at once

## PURULENT PERICARDITIS (code No 420 100 2)

This is usually secondary to other infection elsewhere but is at times caused by contamination of a previous pericardial tap

### Treatment

#### A Specific Measures

- 1 Systemic chemotherapeutic agents Treat infection with indicated chemotherapeutic agents (see p 514)
- 2 Interpericardial antibiotics At the time of removal of the fluid instill 50 000 150 000 units of penicillin or the

app oximat top al am unt f st pt may in o ther in  
dic ted ntibiotic into the pericardial sa d pending on  
ganisms found ( ee p 514) nd repe t wh neve a t p is  
perform d Ch m th ape ti ag t sh uld h continued  
as lo g p pulent effusion is p es nt

#### B C i Me en s

- 1 P ra nt als Perform as ne d d t r liev p ure
- 2 Pericardi t my If fluid is e psulated or pati t is not  
spending to therapy surgical draining m y be n ess ry

### CHRONIC CONSTRICTIVE PERICARDITIS

(code No 420 4)

(Tuberculous pericarditis code No 420 123 4)

This is due to t b culo p i rditis in most of the ses  
in th m under the uol gy i unknown a i w a s m y follow  
ut onap cific pericarditi o tr umatic po i a ditis

#### T im t

A C i M a To combat a ctes and co gestive f ilure

- 1 Low od um diet
- 2 M lal diur i need d to k ep patient dry { ee p  
204 } May e mbine this with int mittent ammonium  
chloride as in c rdiaç fail
- 3 Digitalis is u lly of litl val e

B S gical Removal of C t i ting Pe i dium This p ocedure  
n f q lly t a pati nt t mal h alth If o gestiv  
phe mena a bronc o th pe te rditi i p ogressiv  
a gical int rvention is th only method of f ing poss bl ure

### NEUROCIRCULATORY ASTHENIA (code No 004 580)

(Da Costa s Syndrome or Effort Syndrome)

Neu oci ulatory thena i a bronc di order of y ung adults  
whi h is c nsidered at p esent to b a p ychiatric di o der it is  
ha teri d by four cardinal symptom dyspnea on H rt palpi  
tations left chest pain and a y f tibility The ymptoms re  
often m re related t the em tional onatio of effo t than to  
the ff rt it elf Examination rev al o clini al findings of heart  
diseas altho gh a hyc rdia is oft n pres t

#### T ime t

A ny both repy d R a ranc The m dical examin tion nd  
the m nn of handling th pati t hav importa t therape tic  
v l e

- 1 M dical examination should be tho ough
- 2 Th patient should be a d that no o ganic di order xists
- 3 P ych th r py Furth and m intensive pay h th repy  
may be f value

#### B C ral M res

- 1 Tre tm t of hyperventilation An acute attak may be  
borted by th dminal tration of 5% co bon dioxide re  
bre thing in a bag or by holding th bre th Do not giv

## 182 Pulmonary Heart Disease

ammonium chloride. It does not relieve symptoms and may precipitate acidosis inasmuch as fixed base has been lost in compensating for the alkalosis.

- 2 Good hygiene with moderation in all activities, a well balanced diet and progressive increase in exercise under supervision and with encouragement.

### Prognosis

The prognosis for survival is good but is often discouragingly poor for relief of symptoms.

## PULMONARY HEART DISEASE (Cor Pulmonale)

*Heart disease secondary to disease of the lung or of the pulmonary arteries. Emphysema, pulmonary fibrosis, silicosis and kyphoscoliosis are common causes of chronic cor pulmonale.*

### Diagnosis

- A Symptoms Symptoms of the underlying pulmonary disease may be present; cough with sputum, dyspnea and often wheezing precede the signs of pulmonary hypertension, right ventricular hypertrophy and failure for many years.

B Signs

- 1 Signs of pulmonary hypertension: Systolic pulsation and murmur in pulmonary area; palpable second sound in pulmonary area; accentuated, closely split second sound; possibly early diastolic murmur of pulmonary insufficiency.
- 2 Signs of right ventricular hypertrophy: Heaving right ventricular impulse in the left parasternal area; weak tapping apical impulse; presystolic gallop in the xiphoid area.
- 3 Signs of right ventricular failure follow, usually with sinus rhythm and with oxygen arterial saturation less than 85%.
- 4 Evidence of central cyanosis and high output state may be present.

### Treatment

- A Specific Measures Appropriate antibiotic therapy for the respiratory infection that so commonly precedes failure in this type of case. The patient may be afebrile.

B General Measures

- 1 Intermittent oxygen therapy, possibly by positive pressure to increase arterial oxygen saturation and to decrease pulmonary arterial pressure. Continuous oxygen therapy should be avoided and the patient closely observed for stupor and coma, since carbon dioxide retention may occur with oxygen therapy in these patients (see p. 145).
- 2 The usual methods of treatment of heart failure should be used (see p. 182): bed rest, restriction of sodium, mercurial diuretics and digitalis. Digitalis may not be effective if there is a high cardiac output state.

## CARDIOVASCULAR SYPHILIS

Cardovascular syphilis may manifest itself as an complicated syphilitic aortitis syphilitic aortitis in sufficient secondary atherosclerosis of the coronary artery of the aorta as a general picture of the involvement of the aorta of the coronary artery

The diagnosis may be supported by a history of syphilis evidence of the disease in the body (especially C N S syphilis) and a positive logical test for syphilis Serological tests are negative in about 20% of cases

### Treatment

#### A Specific Measures

- 1 See treatment of late syphilis (p 440)
- 2 The putrid acidosis which the Henschel reaction are with penicillin it is therefore not indicated in early treatment with iodides or mercurials
- 3 Severe subsequent course of penicillin is advised by some authorities at a minimum annual interval especially if the clinical picture remains positive

#### B General Measures

- 1 Blood is distensible during the treatment with penicillin
- 2 Surgical repair of the coronary has been attempted but is best considered as being in the exploratory stage

## SURGERY IN THE CARDIAC PATIENT

### Particular Hazards of Surgery in the Cardiac Patient

- A The usual hazards of general anesthesia are particularly great in surgery of the heart patient namely shock haemorrhage anoxia thrombembolism and infection
- B The above hazards may precipitate any of the following
  - 1 Coronary insufficiency especially if the patient has coronary disease
  - 2 Cardiac failure
  - 3 Cardiac arrhythmia

### Cardiac Risks of Anesthesia

- A Hypotension following preoperative sedation and induction may precipitate coronary insufficiency and arrhythmia
- B Staggering induction increases the work of the heart
- C Anoxia is particularly serious and may cause coronary insufficiency and increase pulmonary hypertension or arrhythmia

### Special Risks Involved in Partial Cardiac Lesion

- A Rheumatic Heart Disease. With the exception of mitral regurgitation these lesions involve hazards depending on functional status of the lesion
- B Hypertensive Pathosis. As a rule these patients develop atherosclerosis of the coronary arteries
- C Coronary Disease
  - 1 Great risk especially if recent infarction



## 194 The Cardiac Patient and Pregnancy

- 2 About 5% additional hazard
- 3 Postoperative infarction may occur if there is significant fall in blood pressure and coronary insufficiency
- 4 Adams Stokes attacks may occur
- D Syphilitic Cardiovascular Disease Especially if associated with angina this lesion suggests involvement of coronary arteries and sudden death may occur
- E Major Hazards
  - 1 Coronary disease
  - 2 Aortic stenosis especially if angina and syncope are present
  - 3 Syphilitic cardiovascular disease
  - 4 Adams Stokes attacks

### Anesthesia in Cardiac Patients

- A Surgical procedures in cardiac patients require a skilled anesthesiologist using anesthesia with which he is most experienced
- B Adequate oxygenation must be maintained at all times
- C Avoid cyclopropane except for induction because of danger of arrhythmias
- D Induction must be smooth
- E Avoid hypotension and treat promptly if it occurs

### Management of the Surgical Cardiac Patient

- A If the patient has had recent myocardial infarction postpone surgery for 3 to 6 months except in the most urgent cases
- B Postpone surgery for at least 3 weeks after recovery from congestive failure
- C Exercise caution when giving fluids containing sodium (including blood) to avoid producing pulmonary edema
- D Treat anemia prior to surgery
- E Treat malnutrition and avitaminosis especially avitaminosis B prior to surgery

## THE CARDIAC PATIENT AND PREGNANCY

### Status of Patient

If heart disease is present what added risk does pregnancy impose? The following information will assist in making an estimation of likelihood of cardiac failure

- A Functional class prior to pregnancy
- B Age of patient
- C Size of heart
- D Structural lesion of heart
- E Presence of arrhythmias
- F Socio-economic status (e.g. if children are at home or if the patient must work)
- G Intelligence and cooperation of patient (e.g. Can the patient rest? Can she stay on a low sodium diet?)
- H Presence of associated disease

Factors Which May Predispose to Failure in Heart Disease

- A Excessive work
- B Upper respiratory infection and sodium retention
- C Anemia
- D Paroxysmal arrhythmia
- E Excessive sodium intake, e.g. diet soda, bicarbonate in form of lin. plasma or blood
- F Rheumatism
- G Others

Assessment of Risk of Heart Disease in Pregnancy

- A Little or No Functional Impairment. Practically all patients who are asymptomatic or who have only mild symptoms with ordinary activities may be allowed to continue to term under close medical supervision. If they develop more severe symptoms within thirty days should be hospitalized and treated if failure and kept in bed until term.
- B Moderate or Marked Functional Impairment. If the patient has pulmonary edema or develops pulmonary edema or has moderate to marked symptoms with activity, mitral valve lesion should be considered. This has been successfully accomplished postpartum. If the patient does not have an operable lesion, such a patient should be hospitalized treated for a diastolic failure and kept in bed until term.
- C Very Marked Functional Impairment. All patients seen during the first trimester who have symptoms on little or no activity and who do not have an operable cardiac lesion should be aborted because of the high incidence of maternal failure and death in this group of patients.
- D Good should be selected after the second month.

Physiologic Load Which Pregnancy Imposes on the Heart

The work of the heart is increased by about 50% at the beginning of about the third month when the blood volume and cardiac output increase. The placenta acts as an arteriovenous fistula. Cardiac failure may occur at any time from the end of the first trimester up to 2 to 3 weeks before term at which time the likelihood of an unaccountable reason decreases.

Management of Labor

- A Cesarean section holds that vaginal delivery is to be preferred except in those cases in which there is an obstetrical indication for cesarean. Coarctation of the aorta may be the only indication for vaginal delivery because of the danger of rupture of the aorta.
- B The second stage should be made as short as possible using forceps when possible.
- C Ectocesis should probably be avoided because of the increased work of the heart which follows.

## CARDIOVASCULAR DRUGS

### DIGITALIS AND DIGITALIS LIKE PREPARATIONS

#### Action of Digitalis and Digitalis like Preparations

- A** In congestive failure digitalis increases the force of contraction of the myocardium and so increases the efficiency of the heart. Digitalis significantly increases cardiac output, decreases right atrial pressure, decreases the renal venous pressure, and increases excretion of sodium and water and so restores some of the hemodynamic and metabolic alterations of cardiac failure. The increased cardiac output causes a decrease in venous pressure; however, a direct effect on the venomotor system has also been postulated.
- B** In the arrhythmias (especially auricular flutter and fibrillation) digitalis slows conduction between auricle and ventricle and depresses the S-A and A-V nodes both by direct action and by stimulation of the vagus nerve.

#### Principles of Administration

- A** Concept of Digitalis Saturation (Digitalization) Digitalis must be administered initially in large doses in order to achieve tissue saturation and obtain a therapeutic effect. After digitalization has been accomplished, smaller doses representing the amount metabolized and excreted are administered daily as long as indications for digitalis persist (usually for life).
- B** Criteria of Adequate Digitalization Digitalis is administered until a therapeutic effect has been obtained (e.g., relief of congestive failure or slowing of the ventricular rate in auricular fibrillation) or the earliest toxic effect (anorexia) is reached.
1. Congestive failure with normal rhythm
    - a. Diuretic action is adequate and edema fluid is lost
    - b. Cardiac size is decreased as dilatation becomes less
    - c. Venous pressure and circulation time return to normal
    - d. Decrease of heart rate results if increase was due to failure
    - e. Engorged tender liver becomes smaller and non-tender
  2. Auricular fibrillation When the rate is below 80 after exercising patient, one can usually consider the patient adequately digitalized. The following simple exercises are adequate:
    - a. Bed patients: Sit up 5 times
    - b. Ambulatory patients: Hop up and down on 1 foot 8 times
  3. Ecg effects The most characteristic change which digitalis produces in the Ecg is sagging of the ST segment and displacement of the T waves in an opposite direction to main deflection. Later there may be a prolonged PR interval. The ST-T changes can not be used as criteria of digitalis toxicity for the effects appear before saturation is present and persist for 2 to 3 weeks after digitalis has been discontinued. However, the Ecg is often of value in determining whether digitalis had been administered in the past 2-3 weeks and may at times give one an idea of the amount.
- C** Toxic Effects of Digitalis There are no non-toxic digitalis preparations and the difference between the therapeutic and

toxic level i v ry sm ll Th ymptoms of t i ty are a follows

- 1 Slight toxic ty Anor xi
- 2 Moderate toxicity Nausea d vmitting h d che mal ise
- 3 Considerable toxicity D rhea ectopi E ts (e p cially e t i ul ) blu ing of vision c nfusio d so lentation
- 4 G ss to i ty S e d rrb abd minal p ■ h gh deg conduction bl cks and uricul r or v tr cula fibrillati

D The ca dinal p nciples f d gitalis therapy r ma n t e wh the on uses a c ud drug su h as the whole le f or one f the purif d gly o ide of dig t ll Th y all h b o dly simil r pharmacologi c tions diff ing only in th xt nt of the re ti ne p od ced th y ll b h v simila ly q al t ti ly Th s differ n es m y be utili d to dvant g with t atm t of th indiv dual patient ■ ticula ly with re p t to pot cy pe d f tion xt nt of abs rpti and dur ti n f ction

#### Indication and R t f Admini t ti

##### A I d t ion f Admini t ti n of D g tall

- 1 Card c failure left ght r mbined with lth unus bythm or uricular fib illation
- 2 A t i f b llation or flutt with a r pid e t i ular rate
- 3 Sup a t i ul pa ymal t hyc di
- 4 P lo to cardiac v g y pecially mtral v lvulotomy in p t l ts with sinus rhythm that if p rysem l icular fb ill t n ceurs duri g f llowing g ry the v nt leu l r t will ot be ees ly p d
- 5 The p v ti of p rrysemale t ular rhythmies in p t l t in whom quindz E e failed o annot b t l t d

##### B R tes f Admini t ti

- 1 Or l dmin st ion d in all ca wh ever dig t ll s need d a d pa nt al dmin t rati on i ot ind cated
- 2 P e teral admini t tion
  - a Em rg n y dig t ll t ion
    - (1) A te pulmon y d ma o other seve failu Ca tion should be d in g l ing th full dig t ll ing do e tn a ingl inj ■ int v ously unde th e cir um tan e Th d g should b given i wly in d l d d do es
    - (2) Treatme t of su i la bythmies when th ed fo control of the ve tri al at is u g nt
  - b Inability t take dig t ll o ally
    - (1) N v es and vomiting d e t any c u
    - (2) Coma
    - (3) Postope tively

#### M thod of Dig t ll t ion

A U t t d C Wh n the pati t has e i d o digitalis p pe ation in th pr ding I w h s

- 1 Parenteral digitalis tion (need i u g t) **CAUTION** never administ r digitalis preparations I V in full digitalising dose unless one is certain that there has be n no digitalis tak n in the preo ding two weeks Always give I V preparations slowly

## DIGITALIS AND DIGITALIS LIKE PREPARATIONS

Glycoside	Preparation Available	Digitalis Dose	Method of Administration	Speed of Maximum Action	Maintenance Dose
Orobanchin	1 c ampules 0.25 mg (1240 gr)	0.25-0.5 mg (1240-1250 gr)	0.25-0.5 mg (1240-1250 gr) line 1 wly I V flow with an the drug (b low)	12-15 hours	Not used
Cantharidin	2 d d mp 1 e 0.4 and 0.8 mg (1450-1475 gr)	0.4 (16 mg) (140 gr)	0.4 (16 mg) I V flow with an the drug (b low)	2-4 d	13 (0.2-0.4 mg) (1500-1550 gr)
Digitalis	1 and 2 mp 1 0.2 and 0.4 mg (1500-1550 gr)	1.2 mg (6) (150 gr)	0.2 mg (3) I V flow with an the drug (b low)	3-6 d	0.25-0.5 mg (1200-1500 gr)
Digitalis	3 mp 1 0.5 mg (1430 gr)	3.5 mg (3 c) (140 gr)	0.5 mg (3) I V flow with an the drug (b low)	14-21 d	0.25-0.5 mg (1200-1500 gr)
Digitalis	0.03-0.08 and 0.1 Gm (1500-1500 gr)	1.0-1.5 Gm (15-22 lb)	0.5 Gm (10 gr) I V flow with an the drug (b low)	6-8 hours	0.05-0.2 Gm (3/4-30 gr)
Digitalis	0.1-0.2 mg (1500-1500 gr)	1.2 mg (150 gr)	0.1-0.2 mg (1500-1500 gr) I V flow with an the drug (b low)	18-21 days	0.05-0.2 mg (1200-1500 gr)
Digitalis	0.25-0.5 and 1.0 mg (1500-1500 gr)	2.3 mg (150-150 gr)	0.25-0.5 mg (1500-1500 gr) I V flow with an the drug (b low)	14-21 days	0.15-0.50 mg (1400-1520 gr)
Digitalis	0.5 mg (1520 gr)	7.5 mg	0.5 mg (1520 gr) I V flow with an the drug (b low)	2-6 d	0.5-2.5 mg (120-125 gr)

## PARENTERAL

## ORAL

- Set et d g d in d according to rapidity of the tne d d
- h Initi l dos g sched l E pt in ma ked me y do not give the tre ve ged git lizing dose in a single dose A good gen ral le is to giv 1/2 to 2/3 f th ve g digitalizing dos imm diat ly and f llow with r mainder in 2 4 hour Observ ear fully for digit li to lcity (see tabl page 136)
- Addition of o l digitalis At th time that the m f l dose is given p r nt r lly it ead s ble to gi or lly an v rag m intenance dose f th prep rat on us d if it p ti nt i ble to swallow By in tuting th d g lly rly optimum d g tal sti n ca be achi v d a d main tain d from th st t It is n tne ss pt gi the s me d g tal s gly eid orally that w s us d f th in ti l m d cat on ( g may digital ze with l V Lan toside E and gi e d git lis fol um fo mai l nan )
- d C ti must be e r i d A pr vious history f digit li the py i sten d f f ult i obt in beca s th new r prepa ti are i stel s and m y b in tabl t of v l able col m The patient m y b un war f th therapy h h a b n r c lving
- Digitali toxic ty h be n see m p t e to who have d i d or w un ware of h ying r elv d th drug Thi i anoth r e n for avo ding a full digit lizing d in a single inj tio
- e Individ l e e s f ea h p ti nt No d sag sch d le will fit all p ti nt
- 2 R p d l digitali tion (within 24 hou ) A singl ral digit lizing do i v u lly n w ac nce n u and omit ing s c mmo making attm tion of d g e f digit li ti very d f f ult
- M ltipl aid s ar usually quite dequate and rapid in tion In all c s lo m d i s t e p r i s i o n i e qu r d b f e a h d se A digit li tion appr h d the dr g should be topped t th fi st ig sympt m of to i lity (s e p 136)

### Oral Administration of the Digitalis Drugs

U g o y	Drug	How Admin i t red
Mod at	D git lis	0.4 Gm (8 gr) q 8 ho f 3 dos
	Dig t ln	0.4 mg (1/30 gr) q 8 hou f 3 d
	D g n	1.0 mg (1/60 gr) q 8 hour to 3 dos
Int m date	D git ls	0.2 Gm (3 gr) t i d f r 2 d ys
		0.1 Gm (1 1/2 gr) q i d f 3 d y
	D git n	0.4 mg (1/30 gr) t i d f r 2 da
	D g ln	0.5-0.75 mg (1/20-1/16 gr) t i d f 2 day
Lea 1		0.25-0.5 mg (1/240-1/320 gr) o i d for 3 days
	Digit lis	0.3 Gm (1 1/2 gr) t i d f 4-5 d ys
	Dig t ln	0.1 mg (1/600 gr) t i d f 4-5 d ys
	D g	0.25-0.5 mg (1/240-1/320 gr) t i d to 4-5 days

- 3 **Slow digitalization** At times it is desirable to digitalize slowly over the course of a week especially if the patient cannot be closely observed during this period. If this is done any of the preparations can be given in daily doses 2 or 3 times the average maintenance dose for 5-7 days. The total digitalizing dose may be somewhat greater than when rapid digitalization is accomplished. One must individualize and no dose will fit all patients. The patient should be instructed regarding the early toxic symptoms when they occur the drug should be stopped for one day and the patient then given the average maintenance dose.
- II **Partially Treated Cases** If a digitalis preparation has been taken within 2 weeks give a quarter of the estimated digitalizing dose and then give additional digitalis cautiously observing patient's response.

#### Maintenance Dose and Methods

The oral route is preferred in maintaining digitalization. The exact maintenance dose must be determined clinically for each patient. (The table on page 199 gives the average doses.)

### QUINIDINE AND QUININE

Quinidine is the drug of choice in the management of most cardiac arrhythmias. Quinine may be used but is only about 30% as effective as quinidine. Only quinidine will be discussed here.

#### Pharmacology

- A **Action** Knowledge of the pharmacological effects of quinidine is important in order to understand the use of the drug. Quinidine has a variety of actions:

- 1 It increases the refractory period of cardiac muscle
- 2 It slows the rate of auricular and ventricular conduction
- 3 It decreases the excitability of the myocardium
- 4 It reduces vagal tone
- 5 It is a general depressant to smooth muscle

As far as conversion of auricular fibrillation is concerned several of these pharmacologic actions oppose each other; the clinical effect depends on which of these actions predominates.

#### B Clinical Pharmacology

- 1 **Route** Can be used orally I.M. or I.V. as occasion demands. The I.V. route should be used only by those experienced in the use of the drug and in urgent situations.
- 2 **Absorption** Orally quinidine is rapidly absorbed, reaches a peak level in about 2 hours, and is relatively slowly excreted. There is a slow decrement to about 30% of the peak level after 12 hours.
- 3 **Excretion and fate of drug** Only 10-20% of orally administered quinidine is excreted in the urine; the remainder is metabolized in the body.
- 4 **Doses per day** After the same dose of the drug is continued for 5 or 6 doses at 2-hour intervals, no significant rise in blood level occurs with further doses at the same interval.
- 5 **Cumulative effect** With a fixed dose of quinidine is given

4 times a day as in a maintenance dose schedule the blood level rises progressively but more slowly reaching a maximum in about 48 to 72 hours. The maximum blood level is then maintained on less than same as long as this same dose schedule is maintained. If high serum blood level is desired the daily dose must be increased or the interval between doses shortened.

Use of the fact that 30-40% of the peak blood level of quinidine is still present in the serum 4 hours following peak dose of quinidine a fixed dose schedule such as 0.4 Gm (6 gr) every 2 hours for 5 doses can be repeated for several days to produce increasing concentrations of quinidine in the blood.

### Use

Widely different opinion has been expressed by various cardiologists on the indication, dosages and dangers of the use of quinidine. It must be remembered that patients in whom quinidine has been used have organic cardiac disease unpredictable accident occur when quinidine is not given to these individuals. Until recently no satisfactory method of blood quinidine determination has been available therapy therefore was often on an arbitrary rather than a quantitative basis.

#### A. Indication

- 1 Ventricular tachycardia
- 2 Conversion of atrial fibrillation to normal rhythm. Most cardiologists feel that the presence of marked cardiac failure as in organic heart disease and a clinical rheumatism favor contraindication to the use of quinidine.
- 3 Atrial flutter if digoxin fails to produce sinus rhythm.
- 4 Paroxysmal atrial and nodal tachycardia.
- 5 Prevention of recurrent paroxysmal arrhythmias.
- 6 Suppression of frequent premature beats especially following myocardial infarction postoperative states.

#### B. Contraindication

- 1 Idiopathic as manifested by fever, purpura, rash, severe hypotension following the administration of 0.1 Gm.
- 2 Complete heart block. Relative contraindication.
- 3 Bundle branch block. Relative contraindication.
- 4 Thyrotoxicosis.
- 5 Adult hemophilia.
- 6 Severe bleeding disorders.

### Route of Administration

A Oral (Quinidine) If indicated. This is the method of choice when possible. Quinidine is especially indicated.

#### B. Parenteral

- 1 Intramuscular preparations. The intramuscular preparations can be used if the patient is unable to take the medication orally and the situation is not critical.
  - a. Quinidine gluconate 0.8 Gm (12 gr) in 10 cc ampules.
  - b. 20% quinidine sulfate in propylene glycol.
  - c. 15% quinidine hydrochloride dissolved in water and antipyrine.
- 2 Intravenous preparation. An intravenous preparation should



be used only when great urgency requires it and by a physician familiar with the use of the drug. Quinidine gluconate 0.8 Gm (12 gr) in 10 cc ampules can be diluted with 50-100 cc 5% glucose and given slowly I V at 1 cc per minute.

### Toxicity

A Idiosyncrasy (see page 201)

B Toxic Effects

- 1 The myocardial toxicity is the most important and should be specifically looked for when quinidine is used. The earliest effects are seen electrocardiographically:
  - a Prolongation of the QT interval
  - b Prolongation of the QRS interval
  - c Ventricular premature beats or ventricular tachycardia
- 2 Nausea, vomiting, and diarrhea. These are rarely critical but may be sufficiently severe to require cessation of the drug.
- 3 Cinchonism. Tinnitus, vertigo, and headache are usually mild but may be important enough to require stopping the drug.

**Caution.** When the QRS interval becomes more than 50% wider than that seen before treatment, or when runs of ventricular premature beats or ventricular tachycardia occur, quinidine should be immediately stopped. In patients with auricular fibrillation who are converted with quinidine, transient S-A block may occur at the time of conversion and nodal rhythm may be temporarily noted. This has not proved to be of clinical significance. In very rare instances, ventricular tachycardia may progress to ventricular fibrillation and sudden death. Prolongation of the PR interval is occasionally seen for a short time when sinus rhythm follows quinidine conversion of auricular fibrillation. This rarely is serious and usually subsides spontaneously as the smaller maintenance doses of quinidine are employed.

#### 4 Other cardiovascular effects

- a Hypotension may occur when large doses of quinidine are used or if the drug is given parenterally. It rarely is significant with ordinary oral doses.
- b Embolic phenomena. Emboli occur in approximately 1% of patients with chronic auricular fibrillation converted with quinidine. The incidence is higher in untreated auricular fibrillation; in fact, auricular fibrillation with frequent emboli is an important reason to attempt conversion to sinus rhythm. Anticoagulants are advised for 1-2 weeks prior to conversion in these cases to prevent the development of new thrombi in the auricles. The hazard of emboli with quinidine has been exaggerated but must be appreciated and regarded as a calculated risk.

### Procedure for Conversion of an Arrhythmia to Sinus Rhythm

- A The patient should be under constant observation, preferably in the hospital, where frequent examination of pulse, cardiac rates, and electrocardiograms may be taken.
- B A test dose of 1 Gm (1½ gr) has been traditionally used to exclude possible idiosyncrasies. Wait 2 hours.



Preparations and Dose	How Administered	Speed of Action and Duration
Amyl Nitrite U S P B P Pearl contains 0.2 cc (3 M)	Break pearl in cloth inhale p r n	Onset 10 sec Lasts 5-10 min
Glyceryl Trinitrate Tablets U S P B P (Nitroglycerin) 0.3-0.6 mg ( $\frac{1}{200}$ - $\frac{1}{100}$ gr)	1 tablet placed under tongue p r n	Onset 1-2 min Lasts 15-30 min
Pentaerythritol Tetranitrate H N H (Peritrate®) as powder or 10 mg ( $\frac{1}{16}$ gr) tablets	Orally every 4-6 hours	Onset 15-30 min Lasts 4-6 hours
Sodium Nitrite U S P B P 30-60 mg ( $\frac{1}{2}$ -1 gr)	Orally every 3-4 hours	Onset 5-10 min Lasts 1-2 hours
Erythritol Tetranitrate U S P 15-30-60 mg ( $\frac{1}{4}$ - $\frac{1}{2}$ -1 gr)	Orally every 4-6 hours	Onset 15-30 min Lasts 3 hours
Mannitol Hexanitrate Tablets U S P 15-60 mg ( $\frac{1}{4}$ -1 gr)	Orally every 4-6 hours	Onset 15-30 min Lasts 4-6 hours

## XANTHINES

Recent studies with cardiac catheterization and metabolic balance studies have demonstrated that intravenous xanthines increase the cardiac output increase renal blood flow and glomerular filtration rate and enhance the excretion of sodium and water they therefore may be valuable in the treatment of cardiac failure In addition they have been shown to increase the coronary blood flow when used in large doses and may on occasion be helpful in angina pectoris

Preparations

- A Oral. A variety of official preparations are available but a satisfactory one is Aminophylline U S P H P (enteric coated) 0.1-0.2 Gm ( $\frac{1}{2}$ - $\frac{3}{4}$  gr) 4-6 times per day
- B Parenteral. Aminophylline Injection U S P H P 0.25-0.5 Gm ( $\frac{3}{4}$ - $\frac{7}{8}$  gr) I V slowly over a 5 minute period or I M may repeat in 2-4 hours
- C Rectal suppositories containing aminophylline 0.3-0.5 Gm ( $\frac{3}{8}$ - $\frac{1}{2}$  gr) may be valuable in impending attack of cardiac asthma or in nocturnal angina pectoris

## MERCURIAL DIURETICS

The mercurial diuretics act by reducing the tubular reabsorption of sodium and chloride They may be used for edema due to most causes except those associated with impaired renal function They are of great importance in congestive failure Avoid excessive use especially if the patient is on a low sodium diet since

these agents may be administered orally or hypodermically alkalinized (see page 187)

#### Parental Preparation

- 1 Mercurophylline Injection U S P (Mercuroanthin®) 1.2 cc i.v. diluted
- 2 Methyl and Theophylline Injection U S P Injection of Methyl B P (Sylrgan Theophyllin®) 1.2 cc i.v. as diluted
- 3 Merallin Injection U S P (Mercurhydine®) 1.2 cc i.v. diluted
- 4 Mercaptopurine Sodium N N R (Thiome in Sodium®) as prepared as dry powder in vial 1.4 Gm (21 gr) in 10 cc vial 4.2 Gm (63 gr) in 30 cc vial Add distilled water to bring to proper volume and refrigerate. Give 0.5 to 2.0 gm/kg as ordered. May be used i.m.

#### Oral Preparation

Although the preparation are not fully evaluated and may be still in the trial stage, they may still be warranted. Several are orally available.

- 1 Mercurohydine with Ascorbic Acid 1.2 tablet after a very meal
- 2 Chlormerodine N N R (Neohydrix®) 10.3 mg (10 mg Hg) 1 tablet or more daily as needed

### OTHER DIURETICS

The following are valuable in the treatment of edema.

- 1 Ammonium chloride 4.2 Gm (60-90 gr) daily for 3-4 days if followed by a period of similar duration. Useful also as a plethysmographic agent in the treatment of edema.
- 2 Carbonic anhydrase inhibitors. Various preparations such as sulfanilamide and Diamox® have been employed but their usefulness has not been fully determined.

### PROCAINE AMIDE HYDROCHLORIDE N N R (PRONE-tyl®)

Procaine amide is a potent myocardial depressant, arrhythmias and respiratory depression are the chief side effects. It is useful in the treatment of nodal and ventricular arrhythmias. To be effective it must be used topically. The arrhythmias are if not on the myocardial arrhythmias. It is more potent than the effect on the ventricular arrhythmias. Clinical experience is still too limited to state whether procaine amide or quinine is the drug of choice in the treatment of arrhythmias.

#### Dose and Administration

- A Oral Preparation (250 mg per 1 cc) 250 mg to 1 Gm (4-15 gr) orally every 4-6 hours in the recommended dose.
- B Intramuscular Preparation (1 Gm ampul) 1.0 cc diluted. The peak effect occurs within 15-60 minutes and a significant blood level is still present after 6 hours. The blood level is

higher and the decrease is slower in patients with congestive failure and renal insufficiency. Hypotension is infrequent with the intramuscular use of the drug in the above dosage.

**C Intravenous Preparation (1 Gm ampules in 10 cc diluent)**

Can be used for ventricular tachycardia of a severe or urgent nature. The drug should be given very slowly 50-100 mg (3/4-1 1/2 gr) per minute up to a dose of 1 Gm (15 gr) with continuous blood pressure and if possible electrocardiographic control.

**Toxicity**

The same precautionary methods outlined in the sections dealing with quinidine are essential when procaine amide is being used.

**A Severe Hypotension.** This is noted particularly with the parenteral use of procaine amide and may be severe enough to require cessation of the drug. This is why frequent blood pressure demonstrations are necessary while the drug is being given.

**B Conduction Defects.** Prolongation of the QRS interval may occur as with quinidine.

**C Ventricular arrhythmias** may occur as with quinidine.

## Chapter 5

# DISEASES OF THE BLOOD VESSELS

### PERIPHERAL ARTERIAL DISEASE

An important consideration in the management of patient with peripheral arterial disease is the determination of (1) the amount of disability due to spasm and (2) the amount of disability due to occlusion. The apy is a method in each case to distinguish disturbances.

**Differential Diagnosis of Common Peripheral Vascular Diseases**

	Rhythmical Disorder (code No 47x 50)	Thromboangiitis Obliterans (code No 402 930)	Arteriosclerosis Obliterans (code No 460 952)
Sex	70-80% female	97% male	Over 75% male
Age	40-60 years	20-35 years	Over 60 years
Extremities involved	Usually popliteal but may be low	40% popliteal 98% low	Always low arterially popliteal
Symmetry	Symmetrical bilateral	Asymmetrical usually bilateral	Asymmetrical usually bilateral
Peripheral arterial pulsations	Persistent	Absent or diminished	Absent or diminished
Cul-de-sac of finger	Small as at tips of fingers and toes	Variable	Variable
Venous volume (phlebogram)	Absent	Often present	Absent
Circulation in testis	Absent	Present	Usually present

**Degrees of Spasm and Occlusion in Peripheral Vascular Diseases**

Disease	Spasm	Occlusion
Arteriosclerosis obliterans	0 to	+++
Thromboangiitis obliterans (Buerger's disease)	+	++
Rhythmical	++	0 to
Arteriovenous malformation	++	++

## Differentiation of Spasm and Occlusion

	Spasm	Occlusion
Color	Livid cyanosis	Blanched
Moisture	Wet	Dry
Veins	Constricted	Full dilated
Temperature	Cold	Cold
Reaction to vasodilating tests	Extremity becomes warm	Extremity remains cold

Adequate differentiation can usually be made on the basis of the first 3 of the above factors. Peripheral arterial disease usually is a mixture of spasm and occlusion but in many cases one factor is more prominent than the other. Therapy is aimed at correcting the physiological abnormalities whenever possible.

Test for Degree of Arterial Occlusion

A simple technic for evaluating the degree of arterial occlusion in the lower extremities, especially the foot, is the reactive hyperemia and elevation test. The test is particularly useful in evaluating treatment and in determining the prognosis of ulcers of the foot.

A. Technic

1. The patient is placed supine and the brachial blood pressure taken.
2. The toes are raised to 65 cm above the auricular level and observed for blanching. (The auricular level is taken at 7 cm below the junction of the manubrium and the body of the sternum [Angle of Louis]).
3. If no blanching occurs, the feet remain elevated and blood pressure cuffs are inflated just above the ankles to a pressure 50 cm above brachial systolic pressure. The occlusive cuffs are left on for 5 minutes.
4. At the end of that time, with the feet still elevated, the pressure in the cuffs is suddenly released and the feet observed for return of color.
5. If at the end of 1 minute color has not returned, the foot is lowered 5 cm and then lowered 5 cm every 30 seconds until color returns. The level at which color returns is noted.

B. Interpretation

1. If the filling pressure (level at which color returns) is 35 cm or more above the auricle, spontaneous healing of an ulcer will occur or if amputation is necessary through the foot the amputation site will heal.
2. If the filling pressure is under 35 cm, the more extensive procedures (e.g. sympathectomy, endarterectomy) or drug therapy must be done to help raise the pressure.

### CHRONIC OCCLUSIVE ARTERIAL DISEASE (Usually Arteriosclerosis)

Treatment

Primarily conservative but thromboendarterectomy, vascular grafts and sympathectomy are of inestimable value in the properly selected case.

A General Measures

- 1 Cigarette smoking (contributing to atherogenic failure) which may interfere with the blood supply
- 2 Diabetes if present must be vigorously controlled
- 3 Tobacco in any form should probably be prohibited but there is no complete agreement on this point in thromboangiitis obliterans or Buerger's disease where treatment is useless in the patient who continues to smoke
- 4 Alcohol habits in moderation are not contraindicated
- 5 A well balanced nutritious diet should be maintained
- 6 Adequate rest and relaxation avoid fatigue

B Local Measures

- 1 A cold extrusion of the foot and cold do not assist but the
- 2 Fungus infections of the feet must be controlled  
Castellani's dye is preferred by many avoiding Whiff's solution (see page 90)
- 3 Infections of and to the affected extremity must be guarded against. The patient should be given the following instructions:
  - a Soak feet for 10-15 minutes in warm water (not hot water) after cutting nail
  - b Bunions require to be trimmed by a physician or a chiropodist
  - c Skin must be kept soft and pliant by rubbing with lanolin or bland vegetable oil 2-3 times daily
  - d Socks should be changed at least once a day. Preferably use two pairs of socks. Top of another kind. Shoes must be well fitted and have no pressure point

C Special Measures The following may be used in an attempt to increase collateral circulation

- 1 Buerger's exercises may be of value. However, do not use if an infection or wound is present. Individualize the exercises for the patient. Do not restrict and restrain all at once.
  - a of the time
  - b Elevate leg about 45 degrees (support the limb on inverted chair or the wall) until blanching or pain occurs (usually in 1-2 minutes or less)
  - c Next allow the leg to dangle freely for 2-3 minutes until maximal rubor occurs. At the same time the feet are moved downward and upward and then inward and outward. The toes are spread and closed while these movements are being made. Do not hold the foot for more than 10 minutes. If the feet are too painful it may be necessary to limit the exercises. Then place legs and body in a horizontal position for 2 minutes
  - d Repeat this complete routine 3 times a day and have 3-5 cases daily
- 2 Mechanical devices may be used but it is probable that the only reliable device is the oscillating bed
- 3 Venodilator drug (see page 212). These are usually of little or no value and, unless there is abnormal vasoconstriction, may actually be harmful. Blood flow studies show a decrease in the blood supply to the ischemic limbs if the elderly are treated with the height of just mild vasodilation due to drugs



**D Treatment of the Severe Stages of Peripheral Arterial De-compensation****1 Treatment of claudication**

- a Teach patient to walk slowly take short steps and to stop to rest before the pain of claudication is fully developed
- b Correct any ligamentous or arthritic disabilities stretching exercises salicylates

**2 Treatment of rest pain**

- a Have patient sleep with the head of his bed elevated 8-10 inches
- b Limit activities rigidly
- c If edema has developed an oscillating bed or Berger's exercises may be prescribed

**3 Treatment of severe infection or incipient gangrene**

- a Start antibiotics as soon as infection occurs (see page 514)
- b Keep extremity horizontal or lowered never elevated. The oscillating bed may be useful
- c Keep the foot free of dressings
- d Room temperature must be comfortable (70°-80° F)
- e Support bed clothes by use of a cradle over affected limb or by a pillow under bedclothes at the foot of the bed
- f Drain purulent pockets thoroughly but gently. This may be accomplished by covering crusted lesion with a few layers of Vaseline® or Xeroform® gauze for 24 hours then applying saline sponges at room temperature and changing frequently during the next 48 hours. Then dress the lesion with a bacitracin or bacitracin-neomycin ointment and a single layer of Xeroform® gauze for 2-3 days. Reinstitute this treatment when necessary

**E Surgical Measures**

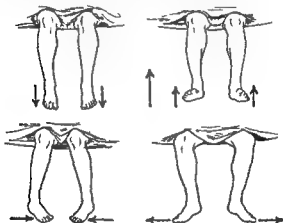
- 1 Thrombo endarterectomy. This procedure is especially useful in the segmental or localized occlusion of major arteries
- 2 Sympathectomy. If there is some evidence of abnormally increased vasomotor tone (see page 208)
- 3 Conservative amputation (toe or transmetatarsal). When reactive hyperemia and elevation test shows a filling pressure in the small blood vessels of 35 cm or more (see page 208)
- 4 Classical supracondylar amputation. If filling pressure in small blood vessels by reactive hyperemia test is less than 35 cm and thrombo endarterectomy or sympathectomy is not indicated

**VASCULAR SPASM****Treatment**

- A General Measures. The same as for occlusive disease. However tobacco in any form must be strictly prohibited
- B Local Measures. The same as for occlusive disease especially if associated occlusion is present
- C Measures aimed at prolonged or permanent relief of spasm
  - 1 Surgery. Sympathectomy of the affected extremity is usually



First position Elevate feet until thoroughly blanched may take 1 to 2 minutes or less



Second position Keep legs dependent until maximal color returns (may take 2 to 5 minutes) Do each series of foot exercises 10 times



Third position Horizontal 2 minutes

the treatment of choice

a Criteria for sympathectomy Best determined on clinical grounds assisted by vasodilator tests

(1) Clinical evidence of increased vasomotor tone This is evidenced by sweating cyanosis and constricted veins (absence of severe rubor and normal or slow blanching reaction on elevation)

(2) Sympathetic block or similar test gives relief of pain, better color to feet and relief of intermittent claudication

b Contraindications Poor venous tone

(1) Marked rubor on dependency

(2) Rapid blanching on elevation

(3) Atrophy of tissues

## 2 Vasodilator drugs

a Chemical sympathectomy The introduction of ganglionic blocking agents has afforded a new approach to the relief of abnormal vasoconstriction Many have been tried but only a few are useful

(1) Adrenergic blocking agents These drugs act at the nerve endings in the vascular muscle cells (neuro-effector site) which is probably the most desirable mode of action They thus not only block the sympathetic vasoconstrictor stimuli but also the vasoconstrictor effects from circulating epinephrine and nor epinephrine This group of drugs includes Benzaxoline (Priscoline®) Phenoxylbenzamine (Dibenzyline®) and Dibenzazepine (Ildar®) (see table) These drugs are effective orally At present they are the most useful in counteracting abnormal vasospasm

Side effects which are troublesome but not serious may be nasal congestion miosis prickly sensation of scalp Weakness dizziness and fatigue which are related to a moderate postural hypotension this may be corrected by a decrease in dosage Overdosage may result in a more profound postural hypotension with faintness or syncope These drugs should be used with caution in any patient who gives a history of asthma or peptic ulcer They may be given intravenously or intra arterially but these routes are rarely necessary

Drug	How Supplied	Dosage
Benzaxoline (Priscoline®)	25 mg tablets	Start with $\frac{1}{2}$ tablet t i d and gradually increase to 4-8 tablets daily
Phenoxylbenzamine (Dibenzyline®)	10 mg capsules	Start with 2 capsules daily and increase by 1 every 4 days up to 4-6 capsules daily
Dibenzazepine (Ildar®)	25 mg tablets	Start with 1 tablet t i d for 1 week then 2 tablets t i d for the 2nd week then may increase to a maximum of 2 tablets q i d

- (2) Vasodilator compounds. Produce peripheral vasodilation by depressing vasomotor activity in the hind brain. They do not block vasoconstricting effects from circulating epinephrine and no epinephrine. They are relatively ineffective in a patient with vascular spasm.
- (3) Intravascular ammonium ion and methanum compounds. Block sympathetic and parasympathetic impulses at ganglionic synapse. They do not block vasoconstricting effect from epinephrine and norepinephrine and may potentiate vasoconstricting responses to epinephrine and norepinephrine.
- (4) Direct vasodilator (acts directly on vascular muscle). Nitrite and nicotinic acid and derivative have not proved to be completely effective in patient with abnormal vasoconstriction.

### ACUTE ARTERIAL OCCLUSION

(Acute Arterial Embolism code No 46 818)

Acute arterial occlusion is usually due to embolism. It occurs most commonly in patients with auricular fibrillation or myocardial infarction but may result from the embolism of vessel especially during periods of hypotension.

The onset is frequently sudden and associated with severe pain. Constitutional symptoms and shock are present if the artery is of large caliber. There is pale, tender of the distal artery and pulseless and coldness of the extremity with numbness, tingling and muscle paresis. If treatment is not instituted the extremity eventually undergoes gangrenous change.

#### Treatment:

A. Surgical removal of embolism is the treatment of choice.

B. Non-surgical Measures. To combat the thrombotic extension of the embolus and relieve vasospasm initiate the following measures: 1. Once and continue postoperatively (if used if urgent relief is not possible).

1. Morphine. Ket 10-15 mg ( $\frac{1}{8}$  to  $\frac{1}{4}$  gr) I.V. at onset and repeat as needed subcutaneously.

2. Anticoagulant therapy should be instituted at onset: 1. p.v. of thrombotic extension of the embolus. Give Heparin Sodium U.S.P. B.P. 2 cc (20 mg) (2000 units) I.V. immediately. The effects of this heparin will usually have worn off by the time the patient has been transported to a hospital or postoperative treatment. The usual regimen of anticoagulant therapy is the standard as soon as possible (see p. 214).

3. Procaine or xylocaine block of the sympathetic system to the affected extremity may be helpful. Repeat as necessary but use cautiously in the patient who has received anticoagulant therapy.

4. Vasodilators and sedatives.

a. Papaverine Hydrochloride U.S.P. B.P. 30-60 mg ( $\frac{1}{2}$  to 1 gr) I.V. every 2-3 hours.

b. Ethyl alcohol (as alcoholic beverage) orally in generous amounts.

® Adrenergic blocking agents (See page 212)

5 Oscillating bed Useful in acute occlusions

**C Local Measures**

1 Keep extremity horizontal or slightly depressed if an oscillating bed is not available protect against pressure or trauma

2 Avoid use of heat or cold to the affected part

**D Treatment of Locking Neuritis** May follow acute arterial occlusion

1 Vitamin B<sub>12</sub> (Cyanocobalamin N N R ) 1000 mcg hypodermically daily for 2 weeks has been advocated

2 Arteriotomy will give relief if Vitamin B<sub>12</sub> therapy does not help but reestablishment of circulation by thromboendarterectomy is preferred

## DISEASES OF THE AORTA

### AORTIC ANEURYSM

(Syphilitic code No 461 147 8)

(Arteriosclerotic code No 461 942 8)

A true aneurysm is a pulsating sac which forms as the result of dilatation of the wall of an artery Those in the proximal part of the aorta especially the arch are most frequently luetic Those farther distal are commonly arteriosclerotic

The signs and symptoms vary with the location and size of the aneurysm Most frequently they are due to local pressure less frequently to rupture The most common symptom is pain which results from pressure on surrounding structures Pressure on structures about the aortic arch also frequently causes dyspnea and cough Abdominal aortic aneurysm may produce back flank or groin pain Some aneurysms may be asymptomatic and may be discovered by physical examination or by x ray of chest or abdomen

#### Treatment

**A Specific Measures** Treat underlying syphilis if present (see page 440)

**B Surgical**

- 1 Replacement of the weakened wall by an autogenous vein-inlay graft homologous arterial graft or Vinyon® or Orion® cloth prosthesis is the preferred operative treatment
- 2 Palliative Attempts to halt further dilatation by producing an internal thrombus along the walls of the sac or by external connective tissue formation are palliative procedures to be used only in the poor risk patient

### DISSECTING ANEURYSM OF AORTA (code No 461 940 1)

Dissecting aneurysm is caused by the rupture of the intima and forceful separation of the coats of the aorta in the presence of hypertension It is usually secondary to arteriosclerotic changes the aorta

It is manifested by sudden onset of severe agonizing pain usually in one of the sites of the rupture. The pain may radiate to the head, back, pelvis, and legs. Shock follows rapidly and death usually occurs in a few hours or days. Diagnosis is usually made at autopsy because of the clinical similarity to myocardial infarction and to arterial occlusion.

### Treatment

Treatment is entirely symptomatic and similar to that of myocardial infarction (see page 165).

## DISEASES OF THE VEINS

### VENOUS THROMBOSIS (code No 48 619)

### THROMBOPHLEBITIS (code No 48 100 7)

A condition of venous thrombology in which a thrombus forms in the vein (usually the lower part of the leg) and grows by deposition of fibrin and by filling the lumen of the leg (98% of cases). Inflammation of a localized area or much of the vein may be present. Early in the disease the chief danger lies in the development of all parts of the thrombus producing pulmonary infarction. Years later the chief danger lies in the development of the postphlebotic leg with edema, subcutaneous fibrosis, and ulceration.

This condition is common in both medical and surgical patients. The present medical and surgical treatment methods may possibly be of value but leave much to be desired.

### Diagnosis

Early diagnosis and immediate therapy is of utmost importance in preventing pulmonary infarction.

#### A History Venous thrombosis is said to occur after abdominal or pelvic surgery, trauma, prolonged bed rest, and in malignancy.

1 Pain in calf and behind knee are important and are usually symptomatic.

2 Pleuritic pain is associated with bloody sputum is highly suggestive of pulmonary infarction.

#### B Physical Examination May be negative

1 Early diagnosis

a Differs in color of feet with elevation

b Slight difference in temperature

c Distention of superficial veins of leg

2 Pain or tenderness on palpation over main venous channels of all feet. Do not palpate too vigorously.

3 Homans sign. Limitation of motion on active dorsiflexion of foot.

4 Swelling of the limb. Usually late sign. May be determined only by measurement and comparison with the opposite limb or by repeated measurements.

5 Examination of the chest in case of suspected pulmonary infarction may reveal signs of diminished breath sounds, crackles, or a pleural friction rub.

Treatment.

**A Anticoagulant Therapy** As soon as the diagnosis of venous thrombosis is made anticoagulant therapy must be started at once *Prothrombin level and Lee-White clotting time must be determined first*

## 1 Heparin

a Intravenous (intermittent) administration If clotting time is normal (5-10 min) give 5-7½ cc (50-75 mg) of Heparin Sodium Injection (Dilute) I S P Injection of Heparin B P every 3-4 hours I V An ideal heparin response is one in which the clotting time is increased 30-60 minutes and returns to normal in 4 hours At least for the first few doses test the clotting time before giving the succeeding dose If the clotting time exceeds 15 min defer the next dose until it falls below this level After checking the clotting time several times it is usually possible to establish a dosage which can be used at 3 to 4 hour intervals It is important not to go too long without adequate therapeutic levels and it is likewise important not to give the next dose when the clotting time is too prolonged

b Subcutaneous Prolonged anticoagulant action of heparin may be obtained by the deposition of a highly concentrated solution of crystalline heparin into a relatively compact and avascular area the subcutaneous fat One injection daily appears to give a prolonged anticoagulant action A highly concentrated aqueous heparin (200 mg per cc) is injected slowly through a No. 25 needle into the subcutaneous fat 1-2 inches below the posterior iliac crest

Average doses are

100 lb patient	200 mg daily
150 lb patient	250 mg daily
175-200 lb patient	250-300 mg daily

Check Lee White clotting time before starting treatment and just before the next dose. Modify dosage as necessary (See previous section)

At present the most general use of heparin is during the first stage or from the first to the third days of anticoagulant therapy until the oral prothrombin depressants become effective The subcutaneous administration of heparin may be used alone without the addition of prothrombin depressants

2 Prothrombin depressants During the first stage of treatment (1-3 days) it is best to supplement these drugs with heparin until prothrombin concentration reaches therapeutic levels (10-30%) Prothrombin levels should be done every day and the next dose not given until the day a level is known

a Dihydroxycoumarin I S P (Dicumarol®) Usually takes 48-72 hours to reach effective therapeutic levels and the same time to return to normal after discontinuing treatment Initial dose is 200-300 mg on the first day 100-200 mg on the second day Maintenance dose varies from 25 to 150 mg daily

b Ethyl Biscoumatate N N R (Tromexan®) Tromexan® is said to induce a more rapid fall in prothrombin

on entation and a m e rapid rise after cessation t  
oral administration than Dicumarol® Initial dose is  
1500 1800 mg in 2 divided doses on the first day and  
300 600 mg on the second day Maintenance dose is  
300 900 mg daily in divid d doses H parin is usually  
only giv n f the first 24 hours because of the m e  
r pid ction f Tromexan®

c Phenindione (Ind n® Heculin® or Danifone®) Has th  
d antage of r pid na t and cessation f action to a d  
g e c mp able with Trom x n® Initial dose is 200  
400 mg in 2 divided doses Maint an e dose is 50 150  
mg in divid d do s Vitamin K is apparently not ef  
fectiv e in counte acting the eff t of ph nindione but this  
m y n t b important in view of the rapid et n to no  
mal p th ombin l is afte topping th dminst tion  
of the drug

3 Duration of th spy Th duration of anticoagulant therapy  
va les with e h se For mo t pati t thi is about 10  
16 days C ntinu the th py fo about 7 day aft r th r  
is no furth r f ve o pan

4 Tr (m nt of bleeding and overd g The p incip l dang  
f om anticoagulant the spy is bn mal bleeding  
a Bleeding d e to ex s h pa in Dis ntinu th the py  
will u ally b i g a ctio f bl d g i bout 1 3  
hour If imm di t tion i ne e y slow l v  
inj tion of protamine sulfate 45 60 mg will d uir li e

b Bl ding du to x ess hshydroxy coumarin (Di umarol®)  
th ff i f s c (50 mg ) of h parin  
or Trom an® This i mor diffic lit ontrol fo th  
p oth ombi lev l rises slowly afte th py i diacon  
thau d Th is is m e ap d wh n Trom an® r  
ph nind on has be n employed

- (1) Sev e b l eding
  - (a) Stop the drug and d not se g in
  - (b) F ash blood (cit at d) t anaf lon imm di t ly
  - (c) Vitamin K<sub>1</sub> em l ion (M phyt n®) 100 200 mg  
I V a l w y ( t r t not o 10 mg /mi te by  
syringe o add d to ve ocly is of d stroas d/  
s lin ) and p i v y 6 hour as nec esa y  
This is t t d to be mo ff tive than yoth ti  
vit min K like p odu t ( m n dione below)
  - (d) M nadiol Sodium Bi ulfit U S P Giv 50 100  
mg I V immediately and epe t 2 3 tim the  
firs d y
- (2) Mild bleeding
  - (a) Stop d ug st rt at low d g n p o
  - (b) Vitamin K<sub>1</sub> (Mephyran®) 50 mg I V above
  - ( ) Men dione Sodi m Bi ulfit U S P Inj ction I  
Menaphthon BP Gt 50 100 mg I V im  
mediately and pe t 2 3 tim th first d y

Overdowag f bi hydr yroumarin (Di uma ol®) o  
Tromexan® with ut bleeding If th prothrombin l i  
d op below 10% and doe of is in 2 days aft  
tinuing bi hydroxy coum in Trom xan® give 50



## 218 Venous Thrombosis

of Mephyton® I V or 20-50 mg of menadione I V  
When prothrombin rises bishydroxycoumarin or  
Tromexan® may again be given

### B Vein Ligation

- 1 If anticoagulant therapy is contraindicated Vein ligation is recommended for any case in which anticoagulant therapy is contraindicated These are cases with purpura open ulcers presence of drainage tubes certain cases of renal or hepatic disease and in cases preparing for C N S surgery
- 2 Active thrombus or embolus formation Vein ligation should be performed if there are signs of propagation of the thrombus if emboli continue to occur while under anticoagulant therapy or if septic phlebitis is present

### C General Measures

- 1 The patient rests in bed with the foot elevated 4-6 inches
- 2 An elastic bandage is applied snugly from the foot ■ above the knee or mid thigh to keep the veins collapsed Do not obstruct arterial circulation Check the pulses Rewrap every 6 hours
- 3 Exercise As soon as treatment is started allow free movement and exercises in bed (see below) If leg is in cast patient may exercise by tensing and relaxing muscles in cast
- 4 Ambulation As soon as the acute pain subsides (or if no pain is present as soon as therapy is instituted) the patient must ■ made ambulatory (unless other systemic conditions prevent this) During this time an elastic bandage should be worn The time out of bed and walking is increased every day The elastic bandage should be worn for about 3 weeks after full ambulation has been achieved

## Prophylaxis

### A Early Ambulation and Exercises

- 1 Early ambulation Prolonged bed rest or inactivity should be avoided especially in elderly patients Have patient up and about as soon as possible after operation or acute illness Walking a few steps is preferable to sitting for half an hour or more in a chair
- 2 Bed exercises If bed rest is necessary passive or active bed exercises should be instituted as soon as possible and should be continued as long as patient must remain in bed These consist of active or passive flexion of toes ankles knee and hips repeated 5-10 times every hour while awake
- 3 Movement in bed With patients at bed rest keep bedclothes loose so patient can move legs freely
- 4 Elevation and compression Elevation of the foot of the bed 4-6 inches and wrapping legs from the toes ■ just below the knees with ace bandages will generally promote venous return

- B Routine Prophylactic Use of Anti coagulants In elderly patients who cannot perform any of the above regimen these may be of value (give as outlined on page 216) but in general the routine prophylactic use of anticoagulants is not advised

## Chapter 9

# DISEASES OF THE BLOOD AND LYMPHATIC SYSTEMS

## ANEMIAS

### HYPOCHROMIC ANEMIA (code No 501.736) (Normocytic and Microcytic)

Hypochromic anemia (low color index and MCH less than 27  $\mu\mu$ ) old anemia and not nutritional deficiency infection and to line and chronic blood loss Women acquire about 4 times as much iron as men with menopause

#### Pathogenesis

A Chronic blood loss

B Inadequate intake of iron (nutritionally anemia)

C Defective absorption of iron in the gastrointestinal tract (e.g. hypochromic anemia of infection) Factors influencing absorption of iron include the following

1 Ascorbic acid facilitates the absorption of iron hydrochloric acid does not

2 Depletion of body iron increases the absorption of iron

3 Vitamin C of ironing steel ( $Fe^{++}$ ) is readily absorbed  $\equiv +++$  (as 1)

4 Infection causes decreased absorption of iron

5 Protein deficiency causes decreased absorption of iron

D Idiopathic mechanism

E Pregnancy

#### Treatment

A Specific

1 Continued ironing content of iron to anemia e.g. hemoglobin deficiency disease omitting iron in the diet may be dangerous if the patient is a child or pregnant

2 Iron preparation of this type of anemia is beneficial given intramuscularly if necessary

a Oral preparations are adequate (Pharmalogical 1 e d is 15 mg/day maximum absorption is considered to be about 100 mg/day)

(1) Ferrous Sulfate USP BP 0.203 Gm (35 g) tid po

(2) Syrup of Ferrous Sulfate NF (0.12 Gm or 2 gr per tsp) tid po (tid po bid o tid po)

SIZE COLOR RELATIONSHIPS OF RED BLOOD CELLS IN THE VARIOUS ANEMIAS

COLOR

NORMOCHROMIC

HYPOCHROMIC

M C H < 21 %  
C I < 0.9

Anemias due to faulty  
GI absorption of iron  
Iron deficiency anemias  
Anemias due to infections  
Anemias due to chemical  
and physical toxins

Splenic anemias  
Erythroblastic anemias  
Anemias due to blood loss

Hemolytic anemias  
Erythroblastic anemias  
Myelophthisic anemias  
Macrocytic anemias  
complicated by iron  
deficiency or blood loss  
or other of the above  
factors

SIZE

MICROCYTIC

M C V < 79 c μ  
V I < 0.9

NORMOCYTIC

M C V 80-94 c μ  
V I 0.9-1.1

MACROCYTIC

M C V > 95 c μ  
V I > 1.1

M C H 27-32 %  
C I 0.9-1.1

Uncommon

As for hypochromic  
microcytic and  
normocytic anemias

Aplastic anemias  
As for hyperchromic  
macrocytic anemias

Uncommon

Uncommon

Pernicious anemias  
Tropical anemias

Anemia of sprue

Plasmodium anemias

Macrocytic anemias

associated with

Idiopathic steatorrhea

Chronic liver disease

Gastric carcinoma (rare)

Faulty ☒ absorption

Leukemia &

Pregnancy

Infancy

- (3) Ferric Carbonate N F 0.3 Gm (5 gr) t i d p  
B P 0.5 1 0 Gm (7½ 15 gr) t i d p c
- (4) Ferrous ammonium citrate solution (50%) 4 cc (1 d )  
t i d p

(5) Ferric Gluconate N F 0.3 Gm (5 gr) t i d p

- b Intravenous iron The patient may fail to respond to iron administered orally because of (a) intolerance due to gastrointestinal irritation (b) resistance to iron (c) severe gastrointestinal disease complicating oral iron (d) refractoriness to oral iron (probably due to inability to absorb the iron)

IV administration of characteristic iron side effects each as provided only the amount necessary to correct the deficiency is given

(1) Calculation Use of the following formula

(a) (Normal Hgb - patient's Hgb) x 0.255 Gm of metallic iron needed

(b) % deficit of Hgb x 25 mg of metallic iron needed

(2) Administration Do not give the total dose at one time administer as follows

(a) Initial dose 50 mg IV

(b) Subsequent doses 100-150 mg IV daily until total dose is given

### 3 Adjunctive therapy

Ascorbic acid Give ascorbic acid or ascorbic acid tablets 30-60 mg per day for children 100-150 mg per day for adults

- b Hydrochloric Acid Diluted USP (10%) 2-4 cc (½-1 dr) t i d in a glass of water with meals sipped through a glass or fibrous food equally has been prescribed for patients with achlorhydria but recent evidence indicates that dilute hydrochloric acid does not facilitate absorption of iron in the gastrointestinal tract. If prescribed the patient should brush the teeth with sodium bicarbonate after meals to neutralize acid left on the teeth

### B General Measures

- 1 Diet High in iron High protein in high iron high vitamin diet At least 70 Gm protein daily for average adult Foods high in iron in liver or other organ meats fresh red meats yeast eggs vegetables especially vegetable greens Poultry prunes prunes A 2240 calorie diet contains 70 Gm protein 115 Gm fat and 230 Gm carbohydrate will contain approximately 20 mg of iron

- 2 Vitamins Vitamin deficiencies are usually multiple and are associated with other nutritional deficiencies. Make a careful survey of nutritional status before administering expensive vitamin preparations which have not indicated indications. Vitamin deficiencies directly or indirectly associated with anemia include beriberi pellagra scurvy and hypoproteinthrombinemia due to vitamin K deficiency

Deficiencies are most often multiple so it is usually advisable to administer multiple vitamin preparations

c Specific preparations Use only specific vitamin

## 221 Pernicious Anemia

deficiency (see page 38)

- 1 Whole blood transfusions preferably of fresh blood are used when there is need for rapid restoration of hemoglobin this need is more urgent when hemoglobin is less than 8 Gm per 100 cc (30%) (see page 247)
  - a Acute hemorrhage when blood loss is greater than 300 cc
  - b Chronic hypochromic anemia when
    - (1) Need for correction of anemia is urgent (e.g. surgery and acute sepsis)
    - (2) Fail re to respond to anti anemic measures Re evaluate and consider other causes for the anemia (e.g. blood dyscrasias and serious constitutional diseases)
- 4 Red cell mass ( sludge ) transfusions are used to restore h moglobin and red cells without
  - a Increasing plasma volum
  - b Producing or incurring risk of serum reactions (serum jaundice)
- 5 Thyroid May be indicated if anemia is associated w th frank hypothyroidism or myxedema (see page 368)

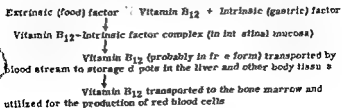
## MACROCYTIC ANEMIAS

### PERNICIOUS ANEMIA (code No 501 702)

Pernicious anemia (P A ) is a chronic and if untreated progressive macrocytic anemia The primary defect is the failure of the gastric mucosa to produce a substance (intrinsic or gastric factor) that is essential for the absorption of the vitamin B<sub>12</sub> in certain foods

In the absence of intrinsic factor in dequate absorption of vitamin B<sub>12</sub> occurs and a deficiency of this vitamin develops Vitamin B<sub>12</sub> is essential for normal erythropoiesis When deficient pathologic red cell formation (megaloblastic regeneration) occurs There is no primary deficiency of folic acid in P A The disease responds specifically to the parenteral administration of vitamin B<sub>12</sub> or of extracts of liver containing vitamin B<sub>12</sub> or to the oral administration of liver or of preparations containing intrinsic factor and vitamin B<sub>12</sub>

The relationship of dietary and gastric factors to normal r b c formation (modification of Castle's theory) may be outlined as follows



## Diagnosis

### A Symptoms

- 1 Anemia Weakness, dyspnea, and palpitation
- 2 Gastrointestinal Anorexia, diarrhea, and dyspepsia
- 3 C N S Numbness and tingling of extremities and sphincter incontinence

**B Signs** Pallor, icteric tint to the face, tachycardia, glossitis, mild hepatomegaly, and splenomegaly; diminution of vibratory sense and reflexes.

### C Laboratory

- 1 Hematocrit achlorhydria
- 2 Macrocytic anemia
  - a MCV  $\geq 100$   $\mu$
  - b MCHC  $> 32$  g% (same as normal)
  - c MCH  $\geq 38$  g% (more)
  - d Orthochromatid megaloblast (normoblasts) present
  - e Anisocytosis, poikilocytosis, and polychromatophilia
- 3 Bone marrow changes
  - a Reticular marrow is increased and soft
  - b Large numbers of megaloblasts are present

### D Therapeutic Response to Vitamin B<sub>12</sub> Extracts of Liver and "LI-55" (Lithium)

- 1 Disappearance of symptoms and signs
- 2 Reticulocyte response (normal count is less than 1%)
- 3 Improvement of anemia Occurs about 1 week after beginning and persists for several months and 48-72 hours after administration of vitamin B<sub>12</sub>

## Treatment

The treatment should be based upon accurate diagnosis. Differentiate from other conditions which can be treated by vitamin B<sub>12</sub>.

**A Special Methods** Paroral therapy is strongly recommended although preparations are available (see below) for those patients who cannot tolerate oral therapy. Patients with severe liver disease or folate deficiency should be administered 5 mg of P.A. daily.

The paroral administration of refined extracts of liver or of vitamin B<sub>12</sub> uniformly followed by optimal clinical and hematologic responses. Following such therapy the P.A. patient in remission will undergo an increase in circulating reticulocytes (reaching a peak in 5-10 days) and a return of erythrocyte and hemoglobin values to normal in 3-5 weeks.

(1) Paroral administration and indications

a For an uncomplicated P.A. in remission

- (1) Initial maintenance 20-30 mg of crystalline vitamin B<sub>12</sub> (Cyanocobalamin) or 1 mg of vitamin B<sub>12</sub> concentrate refined (concentration 1 mg/100 mg) or 1 mg (Liver Injection U.S.P.) Liver Injection contains 10-20 mg of vitamin B<sub>12</sub> per cc giving the equivalent of 20-30 mg of crystalline vitamin B<sub>12</sub>. Vitamin B<sub>12</sub> concentrate may cause allergic reaction.
- (2) Subsequent injections

- (1) Patients in severe relapse Give 10-20 mg of vitamin B<sub>12</sub> or equivalent very first or

until blood values return to normal

(b) Mild relapse in 20 micrograms weekly usually is adequate

- For P A complicated by degeneration of the spinal cord Doses in excess of the amounts needed for uncomplicated P A may be required The degree of reversibility of neurologic manifestations is inversely proportional to the duration of symptoms Improvement frequently is marked in patients with symptoms of 6 months duration or less less pronounced in patients with symptoms of 6-12 months duration and negligible in patients with symptoms of more than 1 year's duration It is advisable to treat all patients intensively for at least 6 months and preferably for 1-2 years

Physical therapy including coordination exercises is an important adjunct to the specific therapy of P A complicated by spinal cord degeneration

(1) Initial I M injection 30-60 micrograms of vitamin B<sub>12</sub> or refined liver extract

(2) Subsequent injections 20-30 micrograms 2 or 3 times weekly for 6 months or more or until optimal neurologic improvement has been demonstrated If optimal neurologic improvement has not occurred at the end of 6 months continue with 20-30 micrograms once a week

- c Maintenance therapy The nutritional requirement for vitamin B<sub>12</sub> in normal individuals is 1-3 microgram or slightly less each day This amount administered to patients with P A in whom blood values have been restored to normal and optimal neurologic recovery has been observed will provide satisfactory control in most instances 15 micrograms of vitamin B<sub>12</sub> or refined liver extract I M once every 2 weeks should be adequate Larger doses may be necessary during periods of increased stress (infection prolonged debilitating or chronic illness etc)

The patient must be instructed as to the need for adequate and regular specific therapy for the remainder of his life The serious risks of neglect should be emphasized

- 2 Oral preparations The response to the oral administration of powdered hogs stomach liver stomach preparations and tablets of vitamin B<sub>12</sub> (even in high dosage) usually is slow or less uniform and often suboptimal when compared with the response to parenteral therapy It is possible that the oral administration of intrinsic factor vitamin B<sub>12</sub> complex substances or the inhalation of powdered vitamin B<sub>12</sub> following intranasal dusting will prove to be satisfactory methods of treatment but the reliability of these methods has not been proved as yet

a Powdered hogs stomach (V ntriculin®)

b Liver with stomach (Extraliv®)

c Tablets of vitamin B<sub>12</sub>

d Powdered vitamin B<sub>12</sub>

e Combinations in tablet form of vitamin B<sub>12</sub> and intrinsic factor (derived from various sources) so called intrinsic factor vitamin B<sub>12</sub> complex substances

B. General Measures Pernicious anemia and liver extract should provide the basis of management as therapy of liver iron and a "weak drug".

1. R. st. Parents with pernicious anemia should be advised that hospital care may be necessary to assist with active involvement (nursing and laboratory examinations).
2. Diet: A diet adequate in calories, minerals and vitamins does not need to be supplemented with extra quantities of dietary live extracts for active treatment. The patient is not on a "weak" diet.
3. Iron: Patients with pernicious anemia may be given an extract to lower the rate when the iron content of the diet is low (M.C.H.C. < 20% or 70 or less index). Liver's patients usually require more iron (see page 215).
4. Hydrochloric Acid: Dose: 1-3 p (1-2 to 3-4 cc) (1-2 to 3 dr) 1-2 in a glass of water with meals. A glass of water with meals may be given to patients who have difficulty as a complication of the achylia. Patients with liver extract will sodium bicarbonate immediately after meals to neutralize hydrochloric acid and prevent erosion of the stomach.
5. Thyroid may be used in patients who will respond due to a so-called hypothyroidism.
6. Measures to improve liver extract in P.A. patients with a so-called hepatic damage have been suggested as an attempt to aid utilization and storage of the extractive action (see page 210).

## MACROCYTTIC ANEMIA OF PREGNANCY (code 43-11-1) (Pernicious Anemia of Pregnancy)

A hyperbromic macrocytic anemia, characterized by megakaryoblasts in the bone marrow usually occurring at end of 1st trimester or during the third trimester of pregnancy.

### Treatment

This anemia responds specifically to the oral or parenteral administration of folic acid or to crude liver extract (which contains folic acid). It does not respond to the parenteral administration of vitamin B<sub>12</sub> or refined liver extract (which contains vitamin B<sub>12</sub> but not folic acid).

- A. Specific Measures After delivery is achieved with folic acid (1 cc a day) (1 cc liver extract) may be discontinued and liver extract may not occur.
1. Folic acid 10-15 mg (1/2 to 1 gr) or 7 daily
  2. Crude liver extract 4 cc (1 dr) 1 M daily
  3. Vitamin B<sub>12</sub> and refined liver extract 1 M daily

### B. General Measures

1. Give liver extract a minimum of animal protein even if diet is already adequate beginning early and continue throughout pregnancy.
2. If hypochromia occurs, iron salts should be administered (see page 210).



## SPRUE (Anemia of Sprue code No 501 703)

Sprue is a chronic disease of undetermined cause (probably due to nutritional deficiency) characterized by sore mouth glossitis indigestion and recurrent diarrhea with steatorrhea it results in anemia asthenia emaciation and even death. The anemia may be microcytic hypochromic normocytic hypochromic or macrocytic hyperchromic (megaloblastic).

### Treatment

#### A. Specific Measures

- 1 For hypochromic anemia Oral or intravenous administration of iron (see page 219)
- 2 For macrocytic hypochromic anemia (with megaloblastosis)
  - a Cortisone or ACTH Cortisone 100 200 mg /day orally or 1 M or adrenocorticotrophic hormone (ACTH) 40 100 units/day 1 M are important advances in the treatment of this form of anemia in non tropical sprue. Improvement in the anemia is thought to be the result of increased absorption from the gastrointestinal tract of nutrients in food including hemopoietic factors (vitamin B<sub>12</sub> folic acid etc.)
  - b Alternate therapy If megaloblastic anemia of sprue fails to respond to cortisone or ACTH therapy give one of the following
    - (1) Vitamin B<sub>12</sub> U.S.P. 15 30 micrograms 1 M 1 2 times per week until remission is obtained and then 10 15 micrograms 1 M every 1 2 weeks
    - (2) Folic acid U.S.P. 10 15 mg ( $\frac{1}{10}$  ¼ gr) daily orally or preferably 1 M
    - (3) Crude liver extract 4 cc (1 dr) 1 M daily

#### B. General Measures

- 1 High caloric high protein low fat high vitamin diet
- 2 Plasma and blood transfusions initially p.r.n. for severe hypoproteinemia and anemia
- 3 Cortisone or ACTH may be used in the hypochromic form in dosages utilized for correction of the megaloblastic anemia of sprue (see page 423). These substances increase the absorption of nitrogen fats and other nutrients from the gastrointestinal tract
- 4 Vitamin K Menadione sodium bisulfite 10 mg ( $\frac{1}{10}$  gr) 1 M or I.V. immediately followed by 5 mg orally twice daily if hypoprothrombinemia is present
- 5 Calcium chloride phosphate or gluconate 2 Gm (30 gr) orally 3 4 times and vitamin D 5000 20 000 units if hypocalcemia or tetany exist
- 6 Vitamin supplements by mouth

### OTHER MACROCYTIC ANEMIAS

This group includes (1) nutritional macrocytic anemia (2) megaloblastic anemia of infancy and (3) megaloblastic anemias secondary to disease of or operative procedures on the gastrointestinal tract

Treatment.

- A Specific Measures Gv folic acid crude liver extract or vitamin B<sub>12</sub> if r prve (see page 225)
- B General Measures Provide an adequate high protein high vitamin diet

**APLASTIC ANEMIA (code No 501.900.0)**

An acute or sometimes chronic disease of the hemopoietic system characterized by an altered production of red blood cells resulting from a depression or exhaustion of the bone marrow. The condition may be secondary to known marrow poisoning but also occur in the primary or idiopathic form.

Diagnosis

- A History of exposure to marrow toxic (e.g. chemicals, certain drugs, and) radiation) is often obtained. Aplastic anemia is a persistent p. grave anemia which like certain other anemias fails to respond to B<sub>12</sub> or iron or di. therapy. Other causes of anemia cannot be demonstrated. Bleeding tendency is common.
- B Laboratory Findings
- 1 Anemia is usually normochromic normocytic
  - 2 Bone marrow often (not invariably) shows aplasia with fatty and fibrous replacement
  - 3 Leukopenia and thrombocytopenia are usually marked

Treatment

- A Specific Measures None are known
- B General Measures
- 1 Transfusions. Repeat administration with careful typing and cross matched whole blood may prolong life in acute periods. Rarely patients who require repeated small amounts of blood transfusions may go into spontaneous remission for a variable time.
  - 2 Discontinue all unnecessary medication.
  - 3 Remove patient from exposure to suspected toxin.
  - 4 Diet. Provide diet with adequate calories, vitamins and minerals.
  - 5 Iron, liver and vitamin therapy. These preparations should be given an adequate trial although therapy of aplastic anemia is unsatisfactory.

**HEMOLYTIC ANEMIAS**Clinical History (Modified after Dameshek)

- A History of Differential Red blood cells show an peculiar morphology and a tendency to hemolysis inherent susceptibility or predisposition to hemolytic reactions
- 1 Spherocytes (familial) anemia (no racial tendency) (see congenital hemolyticicterus code No 513.092)
  - 2 Target cells (Cooley) anemia (Mediterranean group) (familial spherocytic anemia code No 501.997)

3 Sickle cell anemia (Negro race) (code No 513 9x4)

B Acquired Defect (Acquired Hemolytic Icterus) code No 513 911 9) Red blood cells are originally morphologically normal. Etiology includes the following:

- |                                      |   |
|--------------------------------------|---|
| 1 Infections bacterial and protozoal | 4 Immune hemolysins                             |
| 2 Toxins venoms drugs and chemicals  | 5 Agglutinins                                   |
| 3 Physical agents                    | 6 Abnormal splenic mechanisms hypersplenism etc |
|                                      | 7 Certain ovarian cysts                         |

C Unknown Defect (code No 513 900 2)

### Diagnosis

#### A History

- 1 Family or racial hemolytic tendencies (hereditary)
- 2 Exposure to infections toxins agglutinins (acquired)
- 3 Symptoms of anemia (weakness dizziness palpitation and dyspnea) and hemolysis (fever chills abdominal pain and muscle cramps)
- 4 The acute hemolytic crisis is characterized by sudden onset of fever anemia icterus splenomegaly with tenderness and shock

B Physical Examination Pallor icterus tachycardia and fever may be present in all types. Splenomegaly and hepatomegaly occur in the acquired and familial types.

#### C Laboratory Findings

- 1 Increased blood destruction gives rise to
  - a Normocytic anemia
  - b Bilirubinemia
  - c Hemoglobinuria (see table on page 230)
  - d Increased fecal and urinary urobilinogen (urine dark)
- 2 Morphologic r b c defects Spherocytes target cells or sickle cells (see classification above)
- 3 Altered fragility of r b c (always present in hereditary types)
- 4 Increased blood formation is evidenced by bone marrow hyperplasia and presence of immature erythrocytes etc
- 5 Leukopenia sometimes is present in the acquired form

### Treatment of Acute Hemolytic Anemia (Hemolytic Crisis)

Patient must be treated at once Hospitalize whenever possible

#### A Severe Form

- 1 Treat shock (see page 32) and acute anemia. Careful observation of clinical progress is essential
- 2 Whole blood transfusions Blood must be carefully typed (major group and Rh type) and cross matched both at room and body temperatures. Severe reactions may occur even with careful cross matching. Sturgis recommends a cautious preliminary administration of 50 cc of suitable blood followed by an observation period of 1 hour. If no reaction occurs the remainder of the blood may be given over a 2 to 3 hour period
- 3 Plasma If patient cannot tolerate whole blood transfusions (because of hemolysis of injected red cells) plasma transfusions should be administered when necessary to combat shock

- 4 Specific causes should be treated when known
  - a Infections Employ specific anti-infective and supportive measures (see pgs 496 & 514)
  - b Discontinue drugs or remove from contact with poisons or other hemolysins
- 5 Corticotrophin (ACTH) and cortisone may produce striking remissions of the hemolytic reaction and at least temporarily tide the patient over until such time that other more specific measures can be safely instituted. For details of ACTH and cortisone see pag 423
- 6 Splenectomy After shock and fever have subsided and patient's general physical status has improved sufficiently consider if early splenectomy. If hemolytic shock is progressive despite vigorous supportive measures (up to 3 to 4 500 cc transfusions) in young splenectomy may be indicated. When the cause is unknown and reaction has been severe consider for splenectomy after patient has recovered from the hemolytic crisis. Splenectomy is not generally as successful in the acquired form as it is in the familial type and is generally without benefit in sickle cell and familial erythroblastosis anemias
- B Mild Form If the hemolytic reaction is mild only treatment with anti-infective agents and control of opium or colic may be necessary. In the familial type even though the patient is asymptomatic splenectomy may be advisable

# Treatment of Chronic Phases

- A Intractable patient with a mild strenuous relapsing infection exposure to temperature extremes and ingestion or contact with drugs or toxins
- B Splenectomy If patient fails to improve on conservative therapy consider if splenectomy (see above). When abnormal antibodies (iso and auto antibodies) represent the condition splenectomy is often amenable to splenectomy
- C Cobalt chloride 200 mg orally daily has been reported to bring about improvement in the chronic phase of sickle cell and Mediterranean anemias

## MEMOGLOBINURIAS

Diagram 1 (See table on the following page)

### Treatment

- A Specific Measures Remove or treat causative factors
  - 1 Primary malodermis globinuria Treat stage of myphilia present (see pag 440)
  - 2 Fvism Prohibit ingestion of fava beans
- B Symptomatic and Supportive Measures
  - 1 Hemolytic symptoms
    - a Treat acute hemolytic reaction (see pag 328)
    - b Treat chills and muscular aches and pains symptomatic ally
  - 2 Anemia symptoms Treat anemia according to type varying

## DIAGNOSIS OF HEMOGLOBINURIAS

Disease	Precipitated By	Positive Laboratory Tests
<i>Paroxysmal cold hemoglobinuria</i> (code No 510 500)	Chilling or cold	Blood test for syphilis Donath Landsteiner test
<i>Paroxysmal nocturnal hemoglobinuria</i> (code No 510 500)	?	Acid hemolysis test <i>Hemosiderinuria</i> test
<i>Favism</i> (code No 010 3761)	Ingestion of fava beans	None
<i>March hemoglobinuria</i> (code No 510 500)	Exercise	None

Prophylaxis

- A Paroxysmal Cold Hemoglobinuria. Protect against chilling or cold
- B March Hemoglobinuria. Avoid strenuous exercise

## POLYCYTHEMIA VERA (ERYTHREMIA) (code No 501 792)

A chronic disease of the hemopoietic system of unknown etiology characterized by overactivity (erythroblastic) of the bone marrow with resultant overproduction of red cells and hemoglobin. It is manifested by a reddish purple hue to the skin, increased blood volume, capillary engorgement, hemorrhages, venous thrombosis, arterial hypertension, hepatomegaly and splenomegaly, and symptoms referable to multiple organ systems. It is to be differentiated from the polycythemiae that may occur secondarily to known physiological stresses which also cause increased bone marrow activity.

Treatment

- A Definitive Measures. To reduce the total red blood cell volume
- 1 Venesection (phlebotomy)
    - a Utilize careful blood hematocrit determination in following efficacy of treatment
    - b Remove 500 cc. of blood daily until the blood hematocrit reaches a normal level. Subsequently 500 cc. phlebotomies every 2-3 or more months may be sufficient to control mild cases
  - 2 Irradiation inhibition of red cell formation
    - a Radioactive phosphorus ( $P^{32}$ ). This is the most effective anti polycythemic agent available at present. Its use is restricted to institutions equipped to handle radioactive material. It is indicated in patients in which the polycythemia cannot be controlled readily by venesection alone and especially in patients with a history of thrombotic or thrombophlebitic episodes. 4-6 millicuries of  $P^{32}$  (as a phosphate salt) in 2-6 cc. of isotonic (1/2-1 1/2 dr.) sodium phosphate solution are given I.V. If the polycythemia is not controlled following a single I.V. injection, subsequent injections of 3-8 millicuries are given at intervals

of 2 months until the disease is brought under control  
 b X ray irradiation Whole body or spray irradiation may be of benefit when given in repeated dosages. Irradiation of the long bones has proved to be less satisfactory than whole body irradiation in controlling the disease.

### 3 Anti polycythemic drugs

- a Phenyhydrazine hydrochloride or acetylphenylhydrazine  
 Follow patient carefully clinically and with blood studies during and after therapy. These compounds are most safely used if they are administered as maintenance therapy after the hematocrit has been restored to normal by repeated venesections. Give 0.1 to 0.3 Gm (1½ to 5 gr) by mouth weekly as maintenance dose. The use of phenylhydrazine to lower an elevated erythrocyte count, omitting the use of venesection to establish a normal hematocrit and hematocrit level is a hazardous procedure. Anemia is caused and omitting the principal disadvantages of phenylhydrazine therapy.
- b Triethylenemine (TEM) has been employed but experience has been limited.

### 4 General Management

- 1 Provide symptomatic relief as needed.
- 2 Diet: The diet should be adequate and nutritious. There is no rational starvation diet or diet excluding small amounts of blood building food.
- 3 Inform patient regarding the nature of the disease.

- 4 Treatment of Complications: Varies with the status of the polycythemia and the relation of complications to the therapy as well as with the nature and site of the complication. Thrombosis and hemorrhage are common complications.

## ACUTE AGRANULOCYTOSIS (code No 502.7911) (Agranulocytic Angina)

An acute and often fatal usually fatal illness of adult characterised by extreme granulocytopenia which is followed by a fulminating sepsis associated with ulceration of skin and mucous membranes. It is known to be caused by certain drugs and chemicals but is sometimes of unknown origin.

### Differential

#### A History of Medication with Cautious Drug

Sulfonamide	Bismuth	Nitrogen mustard
Aminopyrine	Thiourea and	Methyluracil (7-ar)
Antibiotic	Latex compounds	Alkylating agents
Cinchofen	Gold and other	Tridione
Phenolophen	heptylamine	

- B Physical Examination: Sudden onset of sepsis and fever in inflammation and ulceration of mucous membranes of throat and lips frequently of other areas and of the skin and regional edema.

#### C Laboratory Findings

- 1 Severe leukopenia and granulocytopenia.

## DIFFERENTIAL DIAGNOSIS OF THE LEUKEMIAS AND RELATED DISORDERS

Disease	Duration	Spleno megaly	Hepato megaly	Lymph nodes	WBC		Bone Marrow
					Total Count (usual range)	Differential	
1 Chronic granulocytic leukemia (code No 502 792)	36 mos (8 mos to 16 yrs)	+++	++	±	20 000 500 000	Immature myeloid cells	Myeloid infiltration
2 Chronic lymphocytic leukemia (code No 503 792)	42 mos (8 mos to 8 yrs)	++	+	++	30 000 100 000	Immature lymphoid cells	Lymphocytic infiltration
3 Chronic monocytic leukemia (code No 506 782)		±	±		25 000 100 000 (2 000 500 000)	Immature monocytic cells	Monocytic infiltration
4 Acute leukemias (code No 50 7921)	8 wks (2 wks to 6 mos)	±	±	±	15 000 30 000 (2 000 100 000)	Blast cells (often un differentiated)	Leukemic infiltration Blast cells may be difficult to differentiate
5 Aleukemic myeloid (code No 508 7923)		+	±	±	4 000 (1 000 5 000)	Immature cells (few)	Leukemic infiltration (not always)
6 Agnogenic myeloid metaplasia of spleen (code No 520 958)	11 yrs	+	±	±	20 000 50 000	Immature myeloid cells Nucleated r b c	Normal aplastic or hyperplastic marrow

Not The anemia associated with the leukemias is usually normocytic and may vary from mild to severe. The platelet count is usually increased in chronic granulocytic leukemia but may be decreased in all other leukemias. The platelet count is usually decreased in acute leukemia.

- (2) Parenteral administration of leucovorin (synthetic Leucovorin chloroform factor) in a ratio of leucovorin to antigonat of 1:1:10:1
  - (3) Blood transfusion - Spaced at intervals to maintain RBC at 2.5-3.5 million/cu mm
  - (4) Antibiotics - If infection develops (use appropriate antibiotic) after causative organism has been isolated and sensitivity tests performed
2. Purine antagonist - The antagonist only recently introduced and still in an investigational stage of development but in general thought to be less toxic than the follic acid antagonist
- a. Agent available for use
    - (1) 6-mercaptopurine (Purinethol® 6-MP)
    - (2) 6-thioguanine
    - (3) 6-Diaacetyl 1-beta-rine (azacitidine)
  - b. Indications for use - Acute leukemia, especially in adults. An initial remission rate of approximately 35% has been reported in adults and 50% in children following treatment with 6-mercaptopurine. The status of thioguanine and azacitidine remains undetermined at present
  - c. Dosage and procedure
    - (1) 6-mercaptopurine (6-MP) 2.5-4.0 mg/Kg body weight/day
    - (2) Thioguanine 2.5 mg/Kg body weight/day
    - (3) Azacitidine 2.0 mg/Kg body weight/day

These compounds are administered orally in a single dose or in a divided dose twice daily. The range between the therapeutic and the toxic dose is wider than it is with the follic acid antagonist and hence purine antagonists appear to be somewhat safer to use. 6-mercaptopurine is the drug of choice for initial therapy. It is administered daily regardless of the development of pronounced degrees of leukopenia and neutropenia or pancytopenia until blast (stem) cell polymorphocytes, promyelocytes or promonocytes virtually disappear from the bone marrow or until toxic symptoms develop (see below). Treatment then is discontinued and the patient is watched closely. If complete clinical and hematological remission occurs no further treatment is given until relapse occurs. If no remission or an incomplete remission is obtained the patient is placed on maintenance therapy (usually less than 2.5 mg/Kg body weight/day) in cases in which relapse is not expected to develop. In cases in which relapse is variable response sometimes can be obtained by giving thioguanine or azacitidine in combination with 6-MP.

The effectiveness of purine antagonists depends upon the observation of the patient and on serial blood and bone marrow examinations. The bone marrow rather than the peripheral blood is employed as the principal therapeutic guide. During the stage of drug induced bone marrow suppression, properly spaced blood transfusions must be given as a supportive measure.
  - d. Toxicity - Toxic manifestations of mild degree characterized by anorexia and nausea frequently observed



treatment are markedly enlarged lymph nodes especially if causing pressure symptoms anemia due to bone marrow infiltration or extensive leukemic infiltration of viscera skin etc

(1) Triethylene melamine (TEM) dispensed in 1 mg ( $\frac{1}{50}$  gr) and 5 mg ( $\frac{1}{12}$  gr) tablets for oral use. This relatively new drug appears to be the agent of choice because of its pronounced destructive effect on the mature lymphocyte. *See with caution* especially if the leukocyte count is below 50 000 cells per cu mm. If the leukocyte count is in excess of 50 000 cells per cu mm give 5 mg ( $\frac{1}{12}$  gr) of TEM together with 2 Gm (30 gr.) of sodium bicarbonate orally 1 hour before breakfast (Sodium bicarbonate prevents reaction of TEM with substances in the gastrointestinal tract and permits absorption of the entire dose). On the following day give 2.5 mg ( $\frac{1}{25}$  gr) of TEM plus 1 Gm (15 gr.) of sodium bicarbonate 1 hour before breakfast. Then wait 1 week and check blood counts. Repeat the administration of TEM at weekly intervals reducing the weekly dose to 5 mg ( $\frac{1}{12}$  gr) when the leukocyte count falls below 50 000 cells per cu mm and to 2 to 3 mg as the leukocyte count approaches normal. When normal leukocyte values have been attained discontinue TEM therapy. Remission may last from 6 to 24 months. However during remission blood examinations should be made at intervals of 1 or 2 months.

If the initial leukocyte count is above normal but below 50 000 cells per cu mm give 5 mg ( $\frac{1}{12}$  gr) of TEM or less together with sodium bicarbonate once each week until the desired result is obtained.

(2) Nitrogen mustard (HN<sub>2</sub>) Remissions obtained with nitrogen mustard in chronic lymphocytic leukemia usually are shorter than those obtained with TEM and therefore TEM is preferred to nitrogen mustard therapy. For details of administration of nitrogen mustard see page 241.

b Chronic granulocytic leukemia In contrast to chronic lymphocytic leukemia chronic granulocytic leukemia always should be treated at the time the disease is first discovered. Agents available for treatment are listed in order of preference.

(1) Triethylene melamine (TEM) This agent is quite effective in controlling chronic granulocytic leukemia for long periods of time. It has the advantage of giving remissions lasting from 3 to 10 months but due to the large doses employed it has the disadvantage of causing nausea and vomiting (sometimes severe) in some patients for several hours after administration. However nausea and vomiting may be minimized by administering 25 to 75 mg (3/8 to 1 1/8 gr) of chlorpromazine (Thorazine®) before and at 3 hour intervals after the administration of TEM. It is important to remember that the dosage schedule for TEM in chronic

granulocytic leukemia is significantly higher and therefore quite different from that used in chronic lymphocytic leukemia.

If the leukocyte count is in excess of 100,000 cells per cu mm, give 10 mg ( $\frac{1}{16}$  gr) of TEM and 2.4 Gm (30.60 g) of sodium bicarbonate orally 1 hour before breakfast. On the following day give 5 mg ( $\frac{1}{32}$  gr) of TEM and 2 Gm (30 gr) of sodium bicarbonate 1 hour before breakfast. Repeat the above procedure at weekly intervals after first preliminary blood counts. Reduce weekly dose of TEM when the leukocyte count falls below 100,000 cells per cu mm, and discontinue therapy when leukocyte values approach normal.

If the initial leukocyte count is below 50,000 cells per cu mm, start with 10 mg ( $\frac{1}{16}$  gr) of TEM weekly. If the response to the 10 mg ( $\frac{1}{16}$  gr) dose is unsatisfactory, give 15 to 18 mg ( $\frac{1}{8}$  to  $\frac{1}{4}$  gr) doses of TEM each week.

- (2) Myeleran (GT 41) dispensed in 2 mg ( $\frac{1}{80}$  gr) tablets for oral use. Utilize only in patients having chronic granulocytic leukemia with high leukocyte count. Do not use in patients with normal or subnormal leukocyte counts. Give 4 to 6 mg ( $\frac{1}{10}$  to  $\frac{3}{16}$  gr) daily by mouth until maximum hematologic improvement is achieved, preferably blood counts very severe and on third day. Thrombocytopenia of severe degree may develop on daily oral doses of 10 mg ( $\frac{1}{8}$  gr) or more. When the leukocyte count has returned to a normal level, place in patient on a maintenance dose of 2 to 4 mg ( $\frac{1}{40}$  to  $\frac{1}{10}$  gr) daily. Myeleran is ineffective in acute leukemia and in chronic lymphocytic leukemia.
- (3) 6 mercaptopurina (6 MP) dispensed in 50 mg ( $\frac{3}{4}$  gr) tablets for oral use. Give 3 to 5 mg ( $\frac{1}{20}$  to  $\frac{1}{12}$  gr) /Kg/day in a single or equally divided doses by mouth until the leukocyte count approaches normal. Maintenance therapy (2 to 3 mg /Kg/day or slightly less) then is required to control the disease. 6 MP is ineffective in chronic lymphocytic leukemia.
- (4) Urthane® (ethyl carbamate) dispensed in 0.5 Gm ( $\frac{1}{2}$  gr) plain or enteric-coated tablets for oral use. This compound will control chronic granulocytic leukemia for a relatively long period of time but it has the disadvantage of causing nausea and vomiting in a high proportion of patients and causing anemia. Give 0.5 to 1.0 Gm ( $\frac{1}{2}$  to 1.5 gr) tid until leukocyte count returns to normal, then place on maintenance therapy giving the smallest amount necessary to keep the leukocyte count at or near normal levels.
- (5) Fowl solution (potassium arsenite solution) may be of value when radiation therapy is contraindicated or unavailable.

For technique of administration begins with an initial dose of 0.3 cc (gtt or 5 min) tid orally for 3 days. This dose is increased by 0.05 cc (1 gtt or 1 min) every other day until a dose of 0.8 cc (10

gtt or 10 min) t i d is reached. Further increase of dose 0.05 cc (1 gtt or 1 min) daily until toxic symptoms occur (anorexia, nausea and vomiting, diarrhea) or the leukocyte count approaches normal. Discontinue the drug for 2-5 days and then decrease the maximum dose by 0.05 cc (1 gtt or 1 min) daily until a maintenance level of 0.3-0.5 cc (5 gtt or 5 min to 8 gtt or 8 min) t i d is reached. This dose is continued indefinitely keeping the patient under careful observation.

(6) Nitrogen mustard ( $\text{HN}_2$ ) May produce full clinical remissions in certain early and moderately advanced cases of chronic granulocytic leukemia. These are similar to x-ray response but are of shorter duration. Nitrogen mustard is not recommended for acute leukemias (see page 241).

### B Treatment of Certain Hematologic Abnormalities

1 Anemia Determine whether or not the anemia is myelophthiotic or hemolytic

a Myelophthiotic Treat with the appropriate anti-leukemic chemotherapeutic agent. Adequate nutrition including supplementary vitamins is important but the administration of iron salts is of no value. Periodic transfusions of whole blood may be necessary until the desired chemotherapeutic result has been attained.

b Hemolytic Treat with cortisone or ACTH (see treatment of acquired hemolytic anemia page 228). In the hemolytic anemia of chronic leukemia cannot be controlled by hormone therapy splenectomy may be necessary.

2 Bleeding tendencies Purpuric and hemorrhagic phenomena are often due to the associated thrombocytopenia. Transfusions of fresh whole blood are indicated. Toluidine blue is reported to be of value.

3 Hemolytic crises See page 228.

### C Other Symptomatic Measures

1 Treatment of pruritus See page 67.

2 Treatment of ulcerative stomatitis See page 261.

## **LYMPHOMAS (code No 820) and LYMPHOSARCOMAS (code No 821)**

A large ill defined group of diseases characterized by progressive proliferation of the hematopoietic tissues and manifested by variable involvement of lymph nodes, spleen, bone marrow, liver and other reticuloendothelial structures together with constitutional symptoms of fever, weight loss, hemorrhagic tendencies and anemia. The exact interrelationships of these diseases are not known therefore all classifications remain arbitrary and controversial. Clinical types are often indefinite and may merge into one another.

### Treatment

Certain general principles of management may apply to these diseases as a group.

- A General Measures Measure directed toward maintaining optimum general health should be carried out but they seldom influence the course of the disease per se
- B Radiation and Drugs The effects of radiation and certain chemotherapeutic drugs may be palliative or arresting but are seldom if ever curative. The susceptibility to a particular therapeutic agent and the duration of effectiveness (for both palliation and arrest) will vary not only with each disease but also with the stage of the disease previous to therapy and from patient to patient. The table on page 236 outlines the response of the various hematopoietic disorders to radiation therapy and chemotherapeutic agents.

Although clinical experience has shown that certain of these conditions are more amenable to therapy than others, finally a situation must exist upon a trial of the therapy.

### HODGKIN'S DISEASE (code No 850.954)

A progressive and invariably fatal reticuloendothelial granulomatous (lymphomatous?) disease of unknown etiology involving the lymphoid tissues of the body. It is manifested by progressive enlargement of lymph node, spleen and other lymphoid structures and constitutional symptoms of fever, weight loss and anemia. The lesion can involve any and all tissues; therefore the manifestations are protean. Several clinical and pathological types are recognized, ranging from usually more benign forms, prolymphoma with a survival time of 3 or more years to a rapidly fatal form, sarcoma, with a survival time of less than 1 year. The diagnosis arrived at on clinical grounds must be confirmed by biopsy to differentiate the condition from the chronic infectious granulomas and from the other lymphomas.

#### Treatment

- A Palliative Measures. (No known specific therapy is available.)
- 1 Irradiation. At present, local or total body x-irradiation probably is the palliative measure of choice. Clinical improvement is attributable to regression in the site of involved lymphatic structure and in no way represents a cure of the disease. The average survival time is probably unchanged, but the patient is made more comfortable. Unfortunately, it soon becomes progressively refractory after subsequent courses of ray therapy. Nitrogen mustard therapy should be tried on radio-resistant patients.
  - 2 Combined radiation and nitrogen mustard therapy may occasionally achieve benefits unattainable by either method used alone.
  - 3 Nitrogen mustard [methyl bis(2-chloroethyl)amine hydrochloride] ( $\text{HN}_2$ ) at present is the nitrogen mustard most commonly employed. The indications for its use are:
    - a Widely disseminated chronic granulomatous Hodgkin's disease that has become refractory to x-ray therapy.
    - b Chronic granulomatous Hodgkin's disease with visceral involvement (especially lung parenchyma).
    - c Hodgkin's sarcoma failing to respond satisfactorily to

## BLEEDING DISEASES SUMMARY OF DIAGNOSIS AND TREATMENT

Diagnosis	Diagnosis	Diagnosis	Treatment
<p>1. <b>Thrombocytopenic purpura</b></p> <p>2. <b>Idiopathic thrombocytopenic purpura</b></p> <p>3. <b>Thrombotic thrombocytopenic syndrome</b></p> <p>4. <b>Disseminated intravascular coagulation</b></p> <p>5. <b>Hemolytic uremic syndrome</b></p> <p>6. <b>Thrombotic thrombocytopenic syndrome</b></p> <p>7. <b>Thrombotic thrombocytopenic syndrome</b></p> <p>8. <b>Thrombotic thrombocytopenic syndrome</b></p> <p>9. <b>Thrombotic thrombocytopenic syndrome</b></p> <p>10. <b>Thrombotic thrombocytopenic syndrome</b></p>	<p>1. <b>Thrombocytopenic purpura</b></p> <p>2. <b>Idiopathic thrombocytopenic purpura</b></p> <p>3. <b>Thrombotic thrombocytopenic syndrome</b></p> <p>4. <b>Disseminated intravascular coagulation</b></p> <p>5. <b>Hemolytic uremic syndrome</b></p> <p>6. <b>Thrombotic thrombocytopenic syndrome</b></p> <p>7. <b>Thrombotic thrombocytopenic syndrome</b></p> <p>8. <b>Thrombotic thrombocytopenic syndrome</b></p> <p>9. <b>Thrombotic thrombocytopenic syndrome</b></p> <p>10. <b>Thrombotic thrombocytopenic syndrome</b></p>	<p>1. <b>Thrombocytopenic purpura</b></p> <p>2. <b>Idiopathic thrombocytopenic purpura</b></p> <p>3. <b>Thrombotic thrombocytopenic syndrome</b></p> <p>4. <b>Disseminated intravascular coagulation</b></p> <p>5. <b>Hemolytic uremic syndrome</b></p> <p>6. <b>Thrombotic thrombocytopenic syndrome</b></p> <p>7. <b>Thrombotic thrombocytopenic syndrome</b></p> <p>8. <b>Thrombotic thrombocytopenic syndrome</b></p> <p>9. <b>Thrombotic thrombocytopenic syndrome</b></p> <p>10. <b>Thrombotic thrombocytopenic syndrome</b></p>	<p>1. <b>Thrombocytopenic purpura</b></p> <p>2. <b>Idiopathic thrombocytopenic purpura</b></p> <p>3. <b>Thrombotic thrombocytopenic syndrome</b></p> <p>4. <b>Disseminated intravascular coagulation</b></p> <p>5. <b>Hemolytic uremic syndrome</b></p> <p>6. <b>Thrombotic thrombocytopenic syndrome</b></p> <p>7. <b>Thrombotic thrombocytopenic syndrome</b></p> <p>8. <b>Thrombotic thrombocytopenic syndrome</b></p> <p>9. <b>Thrombotic thrombocytopenic syndrome</b></p> <p>10. <b>Thrombotic thrombocytopenic syndrome</b></p>

# Treatment

## A. Protect from Secondary Infection that Aggravates Bleeding (Toxemia)

- 1 Limit activities. Advise occupations, sports, or other activities which involve minimal physical hazards
- 2 Protect areas of body which are subject to injury
  - a. Lubricate nostrils and other superficial bleeding sites with petroleum jelly to prevent drying and cracking of scabs
  - b. Apply proper elastic bandages, splints, or casts to existing wounds to prevent further potential hemorrhage
  - c. Bandage lower extremities carefully to supply support to surface blood vessels when indicated
- 3 Surgical procedure. Limit the number and extent of operative procedures to a minimum
  - a. Consider need for elective surgery carefully
  - b. Minimize trauma, extent, and duration of operative procedures
  - c. Perform operative procedure in stages (e.g., extract one tooth or remove one toenail at a time)
  - d. Prepare patient for surgical procedures by appropriate hemostatic techniques (e.g., preoperative fresh whole blood transfusion or vitamin K)
- 4 Correct intrinsic factors
  - a. Treat cardiac failure or hypertension when present
  - b. Correct symptoms of violent coughing, sneezing, etc.

## B. Local Bleeding Must Be Treated Promptly

- 1 Bandaging and pressure dressings for hemostasis
- 2 Topical thrombin may be applied locally for hemostasis
- 3 Thromboplastin. Less effective than thrombin
- 4 Absorbable hemostatic dressing (Gelfoam<sup>®</sup>, Oxycel<sup>®</sup>, Fibrinloam<sup>®</sup>)
- 5 Electrical coagulation
- 6 Chemical cauterization. Usually of value only for small bleeding sites, e.g., epistaxis (see page 112). Use tri-chloroacetic acid, ferric chloride, tannic acid, or chrome bead.
- 7 Snake venom (Russell Viper) 1 to 000

## C. Combat Major Hemorrhage or Gross Head Bleeding. Must be treated by measures which combat shock, control bleeding, and correct anemia.

- 1 Combat shock (see page 11). Fresh whole blood (not older than 3-4 hours) is preferred because of its hemostatic as well as its antishock and antianemic effects. Plasma may be used when whole blood is not available.
- 2 Control bleeding
  - a. Blood and derivatives
    - (1) Fresh whole blood transfusions 1 V per n. (see page under Shock). The platelets present in refrigerated whole blood deteriorate largely within 12 hours, although the plasma hemostatic effect may persist for much longer periods. Blood transfusion, carefully administered, should be considered as an emergency supportive measure in all forms of hemorrhage regardless of cause.
    - (2) Plasma transfusion. Transfusion of fresh or frozen plasma (not older than 10 days) provides prothrombin,

fibrinogen antihemophilic globulin and certain other factors which may be of value in controlling bleeding. There are no platelets in plasma.

- (3) Antihemophilic Globulin U S F (Cohn Fraction I) in a single dose of 200 mg (sometimes up to 600 mg may be required) in 5 ml cc physiological saline causes a decrease in the spontaneous clotting time of the blood of hemophilic patients.

b. Vitamins

(1) Vitamin C (ascorbic acid) (See page 83)

(2) Vitamin K (menadione) (See page 80)

(3) Vitamin K and related compounds. Although experimentally this group of agents has been reported to be capable of increasing the capillary resistance in certain disease states in human beings, clinical studies have been generally discouraging. Two preparations available are:

(a) Rutin (N C A) 20-50 mg q i d

(b) Hesperidin Chalcone (N C A) 50 mg q i d

- c. Corticotropin (ACTH) and cortisone may produce striking remissions of the purpuric or hemorrhagic reaction (increased red cell count and platelets and decreased bleeding tendency) and at least tide the patient over until such time as other more specific measures (e.g. transfusions, surgery) can be safely instituted. Dosages of corticotropin of 25 mg I M q i d are generally employed in severe cases; larger doses may be indicated (see page 423).

- d. Antiheparin agents. In anaphylactoid shock, secondary thrombopenic purpura, irradiation reactions, nitrogen mustard therapy, leukemia, menorrhagia, and in certain other conditions, heparin or a heparin-like substance is liberated in excess and appears to be responsible for a bleeding tendency (hyperheparinemia). This excess of heparin may at times be counteracted by the use of two agents: protamine sulfate or toluidine blue, which inactivate heparin by forming stable chemical complexes.

- (1) Protamine sulfate 50 mg in 5 cc aqueous solution I M every 4-6 hours until petechiae cease to appear. 150 mg in 250-500 cc of 5% glucose or normal saline given slowly I V is often given at the time of the first I M injection. Protamine titrations (see J A M A 139 1251 1949) may provide an index as to efficacy of treatment.

- (2) Toluidine blue 6-8 mg/Kg body weight dissolved in normal saline given slowly I V (over a 2-hour period) daily for 3 days and followed by 2-4 mg/Kg for 4 additional days. In preparation of the dye solution it must be passed through a Seltz filter for sterilization and removal of larger dye particles. Transient nausea and vomiting, bluish tint to skin, and blue coloration of urine and feces may be encountered.

- e. Splenectomy. Removal of the spleen may be indicated in selected cases of primary thrombocytopenic purpura, and in cases of secondary thrombocytopenia due to certain

splenic disease (hypersplism). Demonstration of megakaryocytic activity by the bone marrow is essential to the proper evaluation of the individual. Splenectomy is advised only in the hypersplenic form of thrombocytopenic purpura (primary or secondary like Gaucher's, Banti's and gastrointestinal diseases of the spleen) but the operation may be indicated in very selected cases of bone marrow megakaryocytic dysfunction. Thrombectomy is not needed. Splenectomy should be made by a trained hematologist.

### Rh FACTOR

(Reaction due to Blood Transfusion code No 010 38x)

When blood containing the Rh factor (from Rh positive donor) is introduced into a person without the factor (Rh negative) the Rh factor acts as an antigen and globulin may develop against it (anti Rh agglutinins). After the agglutinins have developed Rh positive blood from donor is no longer suitable for transfusion purposes since agglutination and hemolysis of the donor cells is likely to occur. This is very fatal and a serious reaction (as with iron group reaction) may very considerably. Rh sensitization is altogether to be avoided by multiple pregnancies in Rh negative women with Rh positive husband.

### Precautions

#### A General Rules

- 1 All blood for transfusion should be Rh typed in addition to conventional blood grouping and then checked with recipient's blood.
- 2 Rh positive individuals may safely receive blood only from Rh positive donors.
- 3 Rh negative individuals may safely receive blood only from Rh negative donors.

#### B Special Rules Never give Rh positive blood to any of the following

- 1 Rh negative individuals who have had previous transfusions
- 2 Rh negative women who have had multiple pregnancies by Rh positive husbands
- 3 Rh negative pregnant women
- 4 Infants with erythroblastosis

### Treatment

See under transfusion reactions on page 246

## BLOOD TRANSFUSION

### Physiological Reaction

Blood is given to

- 1 Increase circulating fluid volume
- 2 Increase oxygen carrying capacity of blood
- 3 Increase in protein concentration
- 4 Increase coagulability of blood
- 5 Increase immune bodies



## 248 Blood Transfusion

### Contraindications

Transfusions must be given carefully in cases of acute pulmonary edema, cardiac failure, nephritis, and pulmonary embolism or infarction.

### Preparation for Blood Transfusions

#### A Typing and Cross matching

1. Determine blood type of recipient. Use known typing sera: Anti A Blood Grouping Serum U S P (or serum from Type B blood) and Anti B Blood Grouping Serum U S P (or serum from Type A blood). Blood type may be determined according to chart below.

Recipient's r b c		Recipient's Type	
Anti B Serum	Anti A Serum	Landsteiner	Moss
+ Agglutination	+ Agglutination	AB	I
± Agglutination	+ Agglutination	A	II
+ Agglutination	0 Agglutination	B	III
0 Agglutination	0 Agglutination	O	IV

2. Donor should always be of the same blood type as recipient. Cross match as indicated below. In emergency situations Type O (Moss IV) blood (universal donor) may be administered to any type. Type AB individuals may receive blood of any type (universal recipient).
3. Always perform direct compatibility test between donor and recipient blood before each transfusion, even if the blood came from a previously compatible donor. This is done by mixing recipient's cells (RC) and donor's serum (DS) on one side of a glass slide, and donor's cells (DC) and recipient's serum (RS) on the other side.

Donor's cells  
+  
Recipient's  
serum



Recipient's  
cells  
+  
Donor's  
serum

The slide is rocked back and forth for 5 minutes and examined with the low power microscope. If there is any agglutination or suggestion of hemolysis, a new donor must be found.

4. Whenever possible, determine the Rh of the recipient. Rh negative recipients should receive only Rh negative blood. Rh positive recipients may receive Rh positive or Rh negative blood in emergencies when no compatible Rh positive blood is available.

#### B Diseases Which May Be Transmitted By Blood Transfusion

1. Syphilis. Donor should always have a serological test for syphilis.
2. Malaria and hepatitis. Blood from a person with a history of malaria or infectious or homologous serum hepatitis should not be used.

Technique of Blood Transfusion

There are two methods for administration of blood (1) Indirect transfusion using modified blood (blood to which anticoagulants have been added and (2) direct transfusion (blood transfused directly by vein without addition of any substance). The first method is now used almost exclusively.

A Indirect Transfusion Using Modified Blood ■ rate is used most frequently with anticoagulant

- 1 Collection of blood. A specially prepared vacuum flask that contains 30 cc (1 2/3 oz) of 3.5% sodium citrate is commonly used to collect 500 cc (1 pt) of blood. The collection apparatus is equipped with a valve that allows the amount of suction to be regulated. This is the technique used in most blood bank.
- 2 Administration. If the blood is taken fresh, it may be administered directly from the vacuum flask. A special trap that contains a fine mesh filter and a drip measure is inserted into the flask. However, since blood often contains small clots that tend to block the filter, a flask with opening at top and bottom is used as the reservoir for the transfusion.
  - a About 250 cc (1 1/2 pt) of saline is placed in the bottom of the flask and the tubing is filled with saline, leaving about 100 cc (3 oz) of saline in the flask. Clear all air from the tubing.
  - b The blood is then passed through a funnel layered with sterile 4 thicknesses of sterile washed surgical gauze.
  - c The blood is then administered as an intravenous infusion as soon as the filtering is complete.
- B Direct Transfusion. This technique uses an apparatus consisting of a large syringe and a smooth working 3 way stopcock. The blood is drawn into the syringe from the donor through stopcock turned and the blood injected directly into recipient.

Precautions in Administration

- A Always administer alkali (5 Gm or 75 gr sodium bicarbonate) orally or 250 cc (1 1/2 pt) M/s sodium lactate before beginning transfusion. Prophylaxis for hemolytic reaction.
- B Never warm blood before administration.
- C A rate rate is 40 to 50 drops per minute or 150 cc (3 oz) per hour. Can be given at maximum rate of 1 cc (15  $\mu$ ) per second.
- D In cases with myocardial insufficiency give about 1 cc (15  $\mu$ ) per minute (12  $\frac{1}{2}$  drops per minute). Never give over 75 cc (1 1/2 oz) in 1 hour except in treatment of shock.

**COMPLICATIONS OF TRANSFUSION**Transfusion Reaction

Transfusion should be stopped immediately if patient complains of chills, generalized tingling sensation, severe anxiety, precordial oppression, pain in back of neck, thorax and lumbar area or sense of fullness of the head.

A Hemolytic Reaction Most severe of all and may be fatal

Symptoms mentioned above usually appear during the transfusion.

or immediately afterward. Hemolytic reactions are almost always caused by incompatibility of blood.

**II Allergic Reactions** Usually occur following transfusion.

- 1 Mild form: Urticaria, angioneurotic edema, and eosinophilia.
- 2 More severe form: Difficulty of breathing, asthmatic attacks, fatal anaphylaxis may occur.

**C Chemical Reactions (Pyrogens)** Most common reaction. Reaction usually occurs 15 minutes to 1 hour after transfusion. Characterized by chills or rigor followed by fever.

**Treatment of Transfusion Reaction**

**A Hemolytic Reactions**

- 1 **Rational** To attempt to prevent the precipitation of acid hematin in the renal tubules. Therefore, alkalization of urine and forcing of fluids is important.
- 2 **Definitive measures**
  - a Give 10 Gm (150 gr) sodium bicarbonate orally at once and every 4 hours. If patient is unable to hold 20 Gm (150-300 gr) sodium bicarbonate (specially prepared see page 24) in 100 cc of distilled water I.V. or 500-1000 cc (1-2 pt) of M/6 sodium lactate I.V. Repeat the dose in 8 hours or sooner if the urine becomes acid.
  - b Collect all urines and examine for hemoglobin. Continue alkalization until no further hemoglobin is present.
  - c Supply fluids orally or by parenteral means to maintain urine volume of at least 1500 cc (1 1/2 qt) per 24 hours as long as renal function is normal. (See acute renal failure page 303.)
  - d In severe or repeated hemolytic reactions where repeated transfusions may be necessary, corticotropin (ACTH) or cortisone is indicated (see page 423).

**II Allergic Reactions** Treat as a general allergic reaction.

- 1 Give 0.2-0.5 cc (3-8 mg) of epinephrine (adrenaline) 1:1000 subcut at once.
- 2 If symptoms persist, may try antihistamines (see page 66).

**C Chemical Reactions**

- 1 During chill, keep patient warm by adding blankets and hot water bottles. This is usually all that is required.
- 2 However, since the differential diagnosis from the allergic reaction is often impossible, give epinephrine (adrenaline) 1:1000 0.2-0.5 cc (3-8 mg) subcut as soon as possible.

## Chapter 10

# DISEASES OF THE GASTROINTESTINAL SYSTEM

## NONSPECIFIC GASTROINTESTINAL SYMPTOMS

### HALITOSIS ( Bad Breath ) (code No 619)

Halitosis can arise in many causes and treatment is directed at removal or correction of the

#### Treatment

A Correct oral hygiene of the hygienic hygiene

B Treat the disease

- 1 Chronic periodontitis
- 2 Dental caries gum infections to the infection etc
- 3 Systemic diseases and toxemia
- 4 Chronic pulmonary disease lung disease
- 5 Gastrointestinal disease of the GI tract
- 6 Metabolic disease with only the subjective complaint of bad breath is present

C Eliminate the odor from the diet

- 1 Odor and taste
- 2 Rich fatty foods if they are the known cause

### SOOR STOMACH (Pyrosis)

Relieve the condition. Consider partially digested food of low roughage to maintain biliary tract

#### Treatment

A Drug

- 1 Antacid. These drugs (see page 264) often effective in relieving the stomach although it is not felt that the relief is obtained necessarily dependent upon the neutralization of the gastric hydrochloric acid
- 2 Sedatives. Antispasmodic medication (see page 265)

B Physical Diet. (See page 32)

### NAUSEA AND VOMITING

(Nausea code No 611) (Vomiting code No 614)

These symptoms may occur singly or concurrently and may be

due to a wide variety of psychic reflex or central causes

A Psychic Causes These are variable and may have either a superficial or deep seated basis

B Reflex Causes Disturbances of various gastrointestinal structures and other viscera are capable of exciting the vomiting center Correction III this type of vomiting is therefore dependent upon removal or alteration of these reflex disturbances

- 1 Irritation inflammation or mechanical disturbances at any level of gastrointestinal tract (from pharynx to rectum)
- 2 Irritating impulses arising in any diseased viscera e.g. cholecystitis
- 3 Disturbances of semi circular canals e.g. seasickness
- 4 Toxic action of cardiac drugs e.g. digitalis

C Central (Vomiting Center) Causes

- 1 Central emetics Emetine apomorphine morphine
- 2 Exogenous and endogenous toxins
- 3 Increased intracranial pressure
- 4 Cerebral hypoxia Cerebral anemia or hemorrhage

### Treatment.

A Acute. Simple acute vomiting such as occurs following dietary indiscretion or as experienced in the morning sickness of early pregnancy may require little or no treatment When necessary treatment consists of prescribing simple tolerated foods and occasionally mild sedative and antispasmodic drugs

B Prolonged Severe or prolonged nausea and vomiting requires careful medical management Specific causes must be corrected or eliminated The following general measures may be utilized as adjuncts to specific medical or surgical measures

1 Fluids and nutrition Maintain hydration and nutrition

Withhold foods by mouth temporarily Administer 5-10% glucose in saline or water I V in quantities sufficient to maintain adequate hydration When oral feedings are resumed commence with dry foods in small quantities e.g. salted crackers Graham crackers etc With morning sickness these foods may best be taken before arising Later change to frequent small feedings of simple palatable foods Hot beverages tea and clear broths and cold beverages iced tea and carbonated liquids (especially ginger ale) are tolerated quite early Avoid lukewarm beverages Always consider patient's food preferences

2 Drugs

a Sedative antispasmodic drugs may be of value (see page 266)

b Ethyl aminobenzoate (benzocaine) 0.2 Gm (3 gr) with phenobarbital 20 mg ( $\frac{1}{2}$  gr) every 4-6 hours p r n

c Chlorobutanol U.S.P. Chlorbutol B.P. 0.3 Gm (3 gr) every 4 hours as needed

d Chlorpromazine hydrochloride (Thorazine®) has been introduced recently for the control of nausea and vomiting due to a wide variety of causes The drug is administered deeply I.M. in doses of 25-50 mg every 4-6 hours p r n or orally in doses of 10-50 mg every 4 to 6 hours p r n The effectiveness of the drug has not been completely established Hepatic damage with jaundice has

been reported in a few instances. The drug is contraindicated in patients who are receiving large doses of CNS depressants.

### 3. Psychotherapy

- Isolation of patient is recommended. Hospitalization may be necessary. Visiting should be restricted.
- Avoid unpleasant psychic stimuli such as strange odors, foul smelling or foul tasting medication, emesis basins or other unattractive objects as well as foods which are improperly prepared or served.
- Place patient in a definite treatment program and let it be known that something is being done. Hard-boiled or brutal technique is to be avoided.
- All important to relieve the psychodynamic causes of the nausea and vomiting but avoid aggressive psychotherapy during the acute phase of the illness.

## HICCUP (Singultus) (code No. 571)

Hiccup, although a common and usually benign symptom, may be a manifestation of any one of many diseases. It is important to rule out a wide variety of possible causes such as CNS disorders, pulmonary and cardiac disorders, gastrointestinal disorders, renal failure, infectious diseases and other diseases.

### Treatment

Treatment of the specific cause may suffice to clear hiccup. However, it is usually necessary to use certain specific measures to provide relief from this symptom. Countless measures have been suggested for bringing up the hyphoid reflex. All the treatment measures may fail and the symptom may be prolonged and severe as to jeopardize the patient's life.

- Simpl. Home Remedies These measures probably act by distracting the patient's attention thereby interrupting contraction (spasm) of the diaphragm. (1) Inhalation of ammonia or of having patient take firm, deep, rapid sips of water (holding breath, sipping ice water, inhaling long fumes, etc.).

### B. Drug and Medical Treatment

- Sedation. Any of the following common sedatives may be effective. Give Phenobarbital Sodium USP (mebital) Sodium B.P. 0.1 Gm (1½ gr) orally or 0.1 Gm (1½ gr) by rectal suppository.
- Anesthetic drug. Local anesthetics such as cocaine may be applied to the nasal mucosa or to the pharynx. General anesthesia may be tried in so-called intractable cases.
- Antispasmodics. Atropine sulfate 0.3 to 0.6 mg (½ to 1 mg) may be given instantaneously.
- Amyl nitrite inhalations may be effective.
- Carbon dioxide. Have patient breathe into a plain paper bag for 3 to 5 minutes or administer 10-15% CO<sub>2</sub> mixture by face mask for 3 to 5 minutes.
- Surgical Measures Various phrenic nerve operations including bilateral phrenicotomy may be indicated in certain extreme cases.

cases which fail to respond to all other measures and which are considered to be a threat to life

### CONSTIPATION (code No 630)

*Eliminate specific causes of constipation first* Rule out colonic or rectal lesions hypometabolism or psychogenic causes Be especially suspicious when there are sudden changes in bowel habits without apparent cause Inadequate fluids and low residue diets may have a constipating effect The following commonly used drugs which the patient may be receiving for an unrelated illness may cause constipation bismuth salts calcium salts aluminum hydroxide gels (Amphojel®) aluminum phosphate gels (Phosphojel®) and iron salts

#### Treatment

##### A Correct Patient's Attitude Toward Elimination

- 1 A daily bowel movement is not essential to normal health or well being There is normally considerable individual variation in the frequency of bowel movements
- 2 So called auto intoxication theories are unfounded
- 3 Constipation particularly for short periods is seldom cause for alarm
- 4 Many symptoms (e.g. lack of 'pep') attributed to constipation have no such relationship
- 5 Periodic purgation serves no tonic purpose

##### B Re-establishment of Regular Evacuation

- 1 The gastro colic reflex should be utilized to optimal advantage by having patient set aside a regular daily period after a meal (preferably breakfast) for a bowel movement even when the urge to defecate is not present This is based physiologically on the primitive reflex wherein distention of the stomach by food sets off a reflex evacuation of the colon Explanation of the reflex evacuation as it occurs in infants after feedings appeals to many patients Emphasize the fact that this normal reflex is perverted or lost by personal habits or social customs
- 2 Sufficient time must be allotted to permit a leisurely performance of the act
  - a Patient may alter his daily schedule to permit more time for bowel movements
  - b Relaxation may be aided by reading a book etc while sitting on the toilet
- 3 Cathartics and enemata should *never* be employed without direct advice or supervision of a physician if patient expects seriously to correct his constipation since these measures interfere with the normal bowel reflexes For psychological reasons if not physiological it is sometimes inadvisable to discontinue such measures suddenly if patient has employed them for a prolonged period of time It may be better to compromise temporarily with intermediate measures of bland laxatives and mild enemata (see next page) Chronic cathartic and enemata addicts often defy all medical measures and their treatment is especially hopeless when

there is a underlying psychiatric disturbance

C Dieting: The diet may be partially modified to satisfy the following requirements (page 52)

- 1 Adequate volume: On constipation is merely due to inadequate fluid intake
- 2 Adequate bulk: The diet does not necessarily imply roughage as bran. Smooth or bland food may be preferred in constipation
- 3 Vegetables: Ulinas is particularly antacid (e.g. lettuce) and/or with its or vegetable may be of value in many cases of chronic constipation especially the so-called "laxative" type
- 4 Adequate fluids: The patient should be encouraged to drink adequate quantities of fluids so that increased water is available in the intestinal tract for passage of the stool

a Slightly reduced fluid intake if the patient is on a diet of food which is daily sufficient

b The temperature of hot water taken a half hour before breakfast may exert a mild laxative effect

D Exercise: Moderate physical exercise adjusted to individual and physical condition. Bed patients may profit by active and passive exercises. Good tonics for the abdominal lining is the important. Constipation physical therapy employed in patients with physical disorders

#### E Medication

- 1 Bland laxatives: These should be exercised temporarily during the bowel training (re-education) program or as a temporary measure in long standing chronic constipation. It is a substitute for a careful bowel training program. They should be withdrawn as soon as the constipation is relieved

Liquid Pepsin USP Liquid Pepsin B (min 100) 15-30 (12-15) 1-2 times daily per os

- b Ag USP BP with min 100 15-30 (12-15) 1-2 times daily per os

Caution: These should be used over prolonged periods. It is not recommended with the use of foodstuffs particularly fatty foods. There is a risk of lipid pneumonia, which must be avoided

Oil: Oil USP BP 15-30 (12-15) 1-2 times daily per os

- d Vegetable oil: e.g. Pyloric Hydrophilic Milled with Doses NNR (Metformin) 1-2 times daily per os

Cascara Sagrada Aromatic Flavour Extract USP: 1-2 of Cascara Sagrada BP 4-8 c (12-15) 1-2 times daily per os

- f Magnesium Hydroxide USP: 1-2 of Magnesium Hydroxide BP (milk of magnesia) 15-30 c (12-15) 1-2 times daily per os

g Sodium Phosphate USP BP (dosed as phosphate) 1-2 Gm (12-15) 1-2 times daily per os before breakfast

- 1 Sedatives (See page 33)



- 7 Reflex from other viscera
- 8 Neurologic disease
- 9 Metabolic disease

Pelvic pathology (extrinsic to GI tract)  
 Tabes dorsalis  
 Hyperthyroidism

### Treatment

- A** Eliminate the specific cause, whenever possible
- B** Correct Physiological Changes Induced by Diarrhea In addition to necessity for control of intestinal hyperperistalsis it is essential that the following secondary or complicating features be treated
- 1 Fluid imbalance (dehydration) (see page 10)
  - 2 Mineral imbalance e.g. hypocalcemia (see page 380)
  - 3 Nutritional disturbances e.g. hypoproteinemia (see page 56) and deficiencies (see pages 58-64)
  - 4 Psychogenic disturbances e.g. fixation on GI tract or anxiety regarding sphincter mishaps in cases of long standing diarrhea

### **C** Diet

- 1 Non irritant foods Many clinicians feel that food should be withheld or that the intake during the first 24 hours should be restricted to liquid foods (See bacillary dysentery page 276 ) During the acute phase of enteritis the only foods which should be taken by mouth are the following very bland items water weak tea rice or barley gruel meat broth precooked cereals toasted bread or soda crackers with butter and soft cooked (not fried) eggs. These foods are usually administered in about that same order as tolerated
- 2 Bland foods (never highly spiced or seasoned) These foods should be incorporated in the diets of patients convalescing from acute diarrhea or with chronic diarrhea. They include in addition to the non irritant foods the following items cereals with milk or cream strained broths and soups bland cheeses fish fowl and meats (not fried) potatoes (not fried) breads milk products eggs and food beverages (not carbonated)
- 3 Avoid Vegetables and fruits (especially raw) fried foods bran whole grain cereals jams jellies preserves syrups and candies pickles relishes and spices coffee carbonated and alcoholic beverages
- 4 Supplementary vitamins The bland diet is a restricted diet and may further increase the vitamin deficiency induced by altered intestinal absorption. Patients with chronic diarrhea should probably receive vitamins in dosages comparable to those used for chronic vitamin deficiency states. Roughly this amount may vary from 4 to 10 times the normal maintenance dose (see page 56)

### **D** Anti diarrheal Agents

- 1 Bismuth preparations These may be used for either acute or chronic diarrhea
  - a Bismuth Subcarbonate U.S.P. B.S. 2 Gm (15-30 gr.) after liquid bowel movements or q.i.d.
  - b Milk of Bismuth N.F. (bismuth hydroxide and bismuth carbonate) 4 cc (1 tsp.) after liquid bowel movements or q.i.d.

- 1  $\frac{1}{2}$  Bismuth subcarbonate II 30 0 3ss i  
 Camphorated tincture of  
 opium (pareg = c) q = ad 120 0 3i  
 Shake well

Sig 4 cc (1 t p) aft liquid bowel movem ts or q i d

- 2 Milk of bismuth and parego ic (eq al amounts of each)  
 m y b substituted fo the abov mixture using th s m  
 dose

- 3  $\frac{1}{2}$  Belladonna extr ct 0 5 gr viies  
 Bi muth subcarbonate  
 C leum l ctate  
 Kaolin II 30 0 3i  
 P ppe mint oil 2 drop qit ii

Sig 4 (1 t p) t i d a c a d h s or after liquid  
 bow l mov ments p r n (modified after Beckus)

- 4 Pectin kaolin compounds Th se are availabl and are use  
 ful mixtures Dose 15 30 c ( $\frac{1}{2}$  i or) t i d a c and  
 h s or aft liquid bow l mo ements p r n

- 5 Albumin Tannate U S P This drug h s be n recomme d d  
 s an adjuv t othe m sur s when di charg s ar profuse  
 Dose 2 Gm (30 gr) t i d a c and h s or after liquid  
 bow l mo ements p r n

- 6 Opiat s Opi tes m t be avoid d in chronic dia rreus and  
 ar p ferably avoided in acute diarrh s unl ss there is in  
 t act ble diarrhea vomiting and coil Alw ys lud the  
 possibility of i ss gi abdominal dis ase befo e ad  
 ministeri g opi te

- a Camphorat d Tin ture of Opi m U S P B P (pare  
 g ri ) 4 8 cc (1 3 dr) after liquid bow l mo m nts  
 p r n or pr sc ibed in combination with bi muth (above)

- b Cod in Ph phat 15 65 mg ( $\frac{1}{4}$  i gr) subcut after  
 liquid bow l mo em ts p r n

- 7 St ong opi t s Morphine and Dilaudid® should be s serv d  
 fo s l ct d p tients with acute se e e diar h who fail  
 to r spond to more conservative m s ure

- a Morphine ulf te 2 15 mg ( $\frac{1}{8}$   $\frac{1}{4}$  gr) subcut afte  
 liquid bowel mov m nts p r n This drug may produce  
 ss s and vomiting

- b Dilaudid® M y be substit t d fo morphine if th unde  
 ss abi sid eff cts of m rphin n to be avoid d.  
 Dose 1 3 mg ( $\frac{1}{32}$   $\frac{1}{20}$  gr) i M aft liquid bow l move  
 m ts p r n

- 8 Anti spasmodic and edative drugs (c pag 266) Th anti  
 spasmodic drugs particularly when s ed in combination with  
 the b rbitur t exert a f vorable and mild antiperistaltic  
 action It may be n cessary at times to administer the var  
 ious bell domes or belladonna like alkaloids to a point f  
 toxicity in o der to achieve th d sir d effect Anti pa  
 smodi s edative drug m y be onsider d the agents of choi e  
 in th treatm nt of chronic di rrh s associ ted with anxi ty  
 tension stat

- 9 Digestant drugs Hydrochloric acid pe cre tin, and bil  
 s its at times give definit non pecifi sll f Wh the  
 is demon t shl defi l cy of the s what nees replac ment  
 the app is more striking (See spe lll dia s }

Treatment

There is no satisfactory treatment for carcinoma of the esophagus

- A Diet Soft or liquid food should be given as tolerated gastrostomy feedings may be given in selected cases
- B Surgical Removal This is reserved for the few who have no demonstrable metastases and are good surgical risks
- C Deep Radiation Therapy This may be employed in selected cases when surgery is not feasible
- D Morphine sulfate or other suitable analgesic agents should be administered as necessary for relief of chronic pain especially in terminal cases (see page 37)

## CARDIOSPASM (code No 641 )

Spasm of the lower end of the esophagus may be due to local or reflex causes Dysphagia epigastric pain and regurgitation of undigested foods are the most common findings X rays reveal dilatation above the site of obstruction

Treatment

- A Soft or liquid food as tolerated
- B Autonomic drugs have been employed with variable and non spectacular results Large doses of parenteral antispasmodics are often ineffective Recent experimental and clinical studies suggest that the sympatholytic agents may be effective (see page 268)
- C Mechanical dilatation of the cardia by graduated bougies may be necessary

## DISEASES OF THE STOMACH AND DUODENUM

## PEPTIC ULCER

(Gastric code No 640 951) (Duodenal code No 651 951)

An acute or chronic ulceration of any portion of the GI tract which may be exposed to the action of acid juices Lesions may occur at any point in the lower esophagus stomach upper duodenum (most common) gastroenterostomy margins and in certain anomalous areas of the GI tract (e g Meckel's diverticulum) The ulceration may be simple or complicated by hemorrhage perforation scarring and obstruction or by malignancy

Diagnosis

May be based upon

A History

- 1 Pain Classically there is postprandial (1-2 hours) or fasting epigastric discomfort burning or pain usually relieved by bland foods and/or alkalies
- 2 Other symptoms Nausea vomiting flatulence distention hematemesis and melena

B Physical Findings Often local tenderness in the epigastrium

**C Laboratory Findings** There may be

- 1 Abundant or excessive free HCl in gastric juice both with and without histamine injection
- 2 Gross or occult blood in stools

**D X ray Evidence of Ulceration** Based upon films and fluoroscopy of GI series. GI activity is usually indicated by presence of niche or irregularity of mucosal contour but sometimes evidence is indirect such as altered peristalsis, pylorospasm, intestinal obstruction or persistent deformity. Repeated GI series may be necessary to demonstrate active ulceration in certain cases.

**E Gastroscopy** Demonstration of ulceration.

**Diagnostic Criteria**

- A It is not desirable to make a diagnosis of peptic ulcer until there has been x-ray or gastroscopic evidence of ulcer.
- B In face of clear cut peptic ulcer history without laboratory confirmation it may be necessary to perform repeated GI series.
- C Malignant disease should be suspected when the following findings are present:
  - 1 Location: The lesion is located in the stomach particularly if in the pyloric region high on the lesser curvature or on the greater curvature.
  - 2 Duration of symptoms is short (no previous symptoms).
  - 3 Age of the patient is more than 40 years.
  - 4 Response to therapy: The failure of medical and x-ray response after not less than 3 and not more than 4 weeks of intensive medical therapy (see below).

**ACUTE PHASE**

**Treatment**

**A. Local Measures**

- 1 Rest (physical and mental). The patient should have 2 or 3 weeks rest from work if possible or preferably in bed. If the home situation is unsatisfactory or unpleasant or if cooperation of the patient is unsatisfactory hospitalization is recommended. If patient's financial resources are limited it may be necessary for him to continue working in the home in such a case it is essential that he be given careful instructions regarding the carrying out of the medical program under the given working conditions. When possible arrangements should be made for rest periods in reduced hours of sleep and for any other factors which need to be modified in the patient's home or working environment.
- 2 Orientation. The patient should be advised as to the chronic recurrent nature as well as to the potentiality of the disease. Do not emphasize cancer as a complication of the disease.
- 3 Psychotherapy. Anxiety producing mechanism should be relieved where possible. It is not usually wise to institute active psychotherapy during the acute phase of the illness (see page 41).
- 4 Alcohol and tobacco must be avoided.

- 5 Avoidance of certain drugs [e.g. corticotropin (ACTH) and cortisone] Recrudescence of symptoms and even perforation and hemorrhage have been observed to occur in patients with peptic ulcer during the course of administration of corticotropin (ACTH) and cortisone. This hazard should always be considered. The mechanism is unknown.

## B Diet

- 1 Sippy diets. A wide variety of diets are available but the Sippy diets or modifications of these are probably the most effective (see page 52). The patient should learn the principles of his diet and should be taught to be careful of his diet for the remainder of his life. Rich spicy hot and coarse particle foods should be excluded from the diet permanently. Regularity of meals and proper mental attitude during meals should be emphasized as essential for successful results from diet. The length of time the patient should remain on each phase of the diet will depend on numerous factors namely:
  - a Severity of symptoms
  - b Treatment situation e.g. Sippy I diet does not meet the nutritional requirements of the hard laborer. Additional food is essential.
  - c Intelligence and cooperativeness of the patient
  - d Response to treatment
- 2 Avoid short cuts. In general most of the short cut or so-called modified methods do not ultimately save the patient time. In many cases they not only fail to provide the necessary relief of symptoms but also actually serve to lengthen the period of convalescence. The psychological importance of a strict dietary regimen in the acute phase is of great importance to peptic ulcer patients especially to new patients since these patients will otherwise fail to recognize the importance of diet in the long term care of their disease. Patients on short cut diets become sloppy and lachadaisical and indifferent to the potentially serious nature of their illness. Unfortunately there is no unanimity of opinion among clinicians regarding the matter of diet in peptic ulcer.
- 3 Protein hydrolysates. The status of protein hydrolysates in the treatment of peptic ulcer is at present unsettled. Reputed advantages of the amino acids are that they have a high buffering activity and relieve hypoproteinemia as well as causing prompt healing of the ulcer. Antacids are not ordinarily employed with the amino acid regimen. The method is stated to be of special value in hemorrhage due to peptic ulcer.
- 4 Restrictions. Meat extracts, bran, raw vegetables and fruits, fried foods, condiments, spices, alcohol, coffee, tea and carbonated beverages.

## C Drug

- 1 Antacids. It is difficult to state which of the many antacids available are most effective since in certain circumstances each of the agents listed below enjoys special advantages. In general adherence to a suitable dietary regimen will decrease the need for excessive and prolonged use of antacids.

All patients on antacid therapy should be watched for diarrhea constipation alkalosis and fecal impaction.

Antacid powders are prescribed on various schedules according to the stage of the diet. During the strictly stage of the Sippy regime the powder is given on alternate hours or half hours during the day and every night if necessary. The interval between powder administrations then be lengthened according to clinical and x-ray evidence of improvement. For more prolonged use the powder is usually administered 2 hours postprandially. Magnesium dioxide is a laxative drug and calcium carbonate tends to produce constipation.

a Magnesium Oxide U.S.P. B.P. and Calcium Carbonate U.S.P. B.P.

■ Magnesium oxide 30.60 0 3 ii

Calcium carbonate q.s. ad 120 0 3iv

Sig. Take  $\frac{1}{2}$  to 1 tsp. in half glass of water as directed. By varying the magnesium oxide in the above powder the laxative constipating effect of the binding agent may be effectively balanced. This powder may be given in alternate doses with the aluminum hydroxide gel (see below).

b ■ Bismuth (magnesium bismuth carbonate) mixture

■ Magnesium oxide 20.60 0 3v ii

Bismuth subcarbonate 20 0 3

Calcium carbonate q.s. ad 120 0 3iv

Sig.  $\frac{1}{2}$  to 1 tsp. in half glass of water as directed. The bismuth is incorporated because of its soothing coating effect. This powder occasionally offers relief where the magnesium oxide calcium carbonate powder fails to relieve.

c Magnesium Trisilicate U.S.P. B.P.  $\frac{1}{2}$  to 1 tsp. in half glass of water as directed. An excellent non-systemic antacid with unusual protective properties.

d Aluminum Hydroxide G.I. U.S.P. (Amphogel® C Amalinal® etc.). This gel has recently enjoyed popular use because of convenience of administration and freedom from induction of alkalosis and because of local soothing protective and demulcent action. However, they are constipating interfere with phosphate and vitamin absorption, may require large doses and occasionally fail to give relief.

(1) Aluminum hydroxide gel, liquid 1 to 2 tsp. in half glass of water every 2 to 4 hours postprandially.

(2) Aluminum hydroxide gel (dry) tablet chew 1 to 2 tablets and follow with half glass of water every 2 to 4 hours postprandially. These tablets are especially convenient for patients who are forced to continue work or to travel.

(3) Aluminum hydroxide gel magnesium trisilicate mixture liquid (Gelucel® T1 emulsion®) et al. 1 to 2 tsp. in half glass of water every 2 to 4 hours postprandially. The addition of magnesium trisilicate increases the neutralizing power and protective coating action of the aluminum hydroxide gel. This mixture is also constipating.

(4) Aluminum hydroxide gel (dried) magnesium trisilicate tablets chew 1 or 2 tablets every 2-4 hours p r n and follow with a half glass of water

- 2 Sedative drugs The use of sedative drugs will depend on the emotional status of the patient. Tense and apprehensive patients will usually profit greatly from proper sedation. Most patients with peptic ulcer profit by sedative drugs. The barbiturates are the preferred sedatives. They may be used alone or in combination with antispasmodic drugs. Hypnotic doses of the barbiturates may be necessary to insure sleep during the acute phase of the ulcer (see page 35)

- 3 Antispasmodic drugs Any of the following drugs or mixtures may be employed for their antispasmodic and sedative effects

a  $\mathcal{R}$  Tincture of belladonna 10 30  $\mathcal{R}$  3iss 3i

Elisir of phenobarbital q s ad 120 0 3iv

Sig 1 tsp in half glass of water t i d 30 30 minutes

a c and h s p r n

b  $\mathcal{R}$  Belladonna extract 3  $\mathcal{R}$  3 mg gr  $\frac{1}{8}$   $\frac{1}{4}$

Phenobarbital 15 mg gr  $\frac{1}{4}$

Sig 1 tablet t i d 20 30 minutes a c and h s p r n

- c Tincture of Belladonna, U S P B P 0 3 0 6 cc (5 10 drops) in half a glass of water orally t i d 20 30 minutes a c and h s p r n (0 6 cc of the tincture equals about 0 2 mg of atropine) This preparation permits rather delicate titration of desired antispasmodic effect by simply regulating the number of drops

- d Belladonna Extract U S P B P 8 15 mg ( $\frac{1}{8}$   $\frac{1}{4}$  gr) tablets or capsules orally t i d 30 30 minutes a c and h s p r n (15 mg equals about 0 2 mg atropine alkaloid)

- e Atropine Sulfate U S P B P 0 4 0 6 mg ( $\frac{1}{150}$   $\frac{1}{100}$  gr) 1 tablet t i d 30 minutes a c and h s p r n

- f Trasentine® (N C A) 75 mg ( $\frac{1}{4}$  gr) t i d 20 30 minutes a c and h s p r n Acts principally on smooth muscles and has few of the side effects of the belladonna preparations but also has less antispasmodic effect. Usually prescribed in combination with phenobarbital. Stated to have a local anesthetic effect on mucous membranes

$\mathcal{R}$  Trasentine® 0 050 gr  $\frac{3}{4}$

Phenobarbital 0 020 gr  $\frac{1}{3}$

Sig 1 tablet t i d 30 minutes a c and h s p r n

- g Methantheline bromide (Banthine®) and the more recently introduced propantheline bromide (Pro-Banthine®) have been reported to provide effective control of hypermotility and hyperacidity in patients with peptic ulcer. Although these are potent anticholinergic drugs adjunctive measures of diet rest and antacids are often necessary for control of symptoms of many ulcer patients. Methantheline is employed in doses of 50 100 mg ( $\frac{3}{4}$   $\frac{1}{2}$  gr) t i d q i d propantheline is given in doses of 15 30 mg ( $\frac{1}{4}$   $\frac{1}{2}$  gr) t i d q i d

- h Diphenmethanil methylsulfate (Prantal®) is reported to exert significant antispasmodic anticholinergic effects in peptic ulcer patients when used in dosages of 100 200 mg

(1½ 3 gr) t i d q i d  
 Oxyphenonium bromide (Antrenyl®) is reported to be a potent anti cholinergic agent for treatment of peptic ulcer. Dosage 10 mg (½ gr) q i d for the first week in the usual dosage thereafter is reduced to the point of control of symptoms.

## CONVALESCENT PHASE

- Time**      **min**      **tion**      When clinical picture of the lesion is evident (based on freedom from symptoms) a repeat GI x ray series is advisable to determine whether or not the ulcer is roentgenologically healing of the ulcer. In the case of gastric lesions failure of clinical improvement and x ray impoement of the ulcer criteria within a period of 3-4 weeks on a successful medical regimen should be taken as a suspicious evidence of gastric malignancy.
- Ed**      **ation**      **of**      **Pain**      **nt**      **R**      **ga**      **ding**      **R**      **cur**      **n**  
 1. The patient has been stated an understanding of the chronicity and recurrent possibilities of his ailment as well as of the danger of complications which may follow neglectful or improper treatment.  
 2. Factors causing recurrence of ulcer should be emphasized to the patient that the following factors most frequently responsible for recurrence of ulcer are:  
 a. Improper diet and irregular eating habits  
 b. Irregular living habits long irregular hours  
 c. Use of alcohol or tobacco  
 d. Emotional stress  
 e. Infection particularly of the upper respiratory tract  
 3. Management plan should be that directed to the removal of symptoms or modification of the patient's exposure to conditions known to aggravate peptic ulcer. In addition to diet modification and other modification should be readily available to the patient.
- C**      **R**      **t**      **and**      **R**      **e**      **tion**  
 1. Retraction variations and mental illness should be considered in the patient's long day history.

## TREATMENT OF COMPLICATIONS

### INTRACTABILITY TO TREATMENT

Although numerous cases undoubtedly exist where benign peptic ulcer fails to heal despite competent medical supervision it is probable that most cases of intractable peptic ulcer are the result of a combination of factors on the part of the patient. The factors previously mentioned as being possible for the occurrence of peptic ulcer are of the same effect which interfere with the healing process.



of ulcers. The designation intractable should be reserved only for those patients who have been given adequate and supervised trial of therapy. The possibility of malignancy or of other complications of the ulcer (e.g. pyloric obstruction, perforation, gastritis, etc.) must always be considered.

## HEMORRHAGE

(Stomach: code No 840 951 T) (Duodenum: code No 851 951 T)

Although peptic ulceration accounts for about 70% of gross hemorrhage from the upper gastrointestinal tract, one must bear in mind the possibility of esophageal varices, gastritis, duodenitis, carcinoma of the stomach, hiatus hernia, and bleeding diseases.

### Treatment

#### A. Emergency Measures for Hemorrhage and Shock

*Refer to page 3, for general management of shock*

- 1 Hospitalize patient at absolute bed rest
- 2 Warmth. Keep patient comfortable. If an ice bag is applied to the epigastrium, avoid chilling the patient.
- 3 Treatment of apprehension and anxiety
  - a Reassurance by word and manner of physician that the condition is not critical
  - b Rest. Provide prompt mental and physical rest; this can best be achieved in the hospital
  - c Sedation. May be necessary
    - (1) Morphine should be avoided if possible, since it may cause nausea. Dose is 12-18 mg ( $\frac{1}{2}$ - $\frac{3}{4}$  gr) subcut every 4-6 hours. It is best to substitute codeine phosphate 30-60 mg ( $\frac{1}{2}$ -1 gr) subcut or orally or Dilaudid® 4 mg ( $\frac{1}{16}$  gr) subcut every 4-6 hours p r n
    - (2) Sodium phenobarbital (sodium phenobarbitone) 0.03-0.1 Gm ( $\frac{1}{2}$ -1  $\frac{1}{2}$  gr) subcut or orally during the first 24-48 hours
    - (3) Phenobarbital (phenobarbitone) may be continued for several days if necessary
- 4 Oxygen. Preferably by mask at 5-10 liters per minute (see page 147)
- 5 Transfusions. There has been considerable controversy regarding the use of blood transfusions in bleeding ulcer. However, it is generally agreed that the previous conservative attitude (fear that transfusion may raise the fallen blood pressure to a point causing recurrence of hemorrhage) is not warranted. Certainly in severe hemorrhage the time, rate and volume of blood administration should suit the physiological needs, and large amounts of blood may be given when indicated. Transfusions must always be given if hemorrhage is severe (Hgb < 50% or RBC < 3 million) if immediate surgery is contemplated, or if symptoms of anoxia or shock are not rapidly controlled. Slow and continuous administration of 500 cc up to 2500 cc of whole blood daily may be necessary.
- 6 Clinical and laboratory studies

- a Take pulse, respirations and blood pressure every  $1\frac{1}{2}$  to 1 hour since the data may give information regarding shock status in advance of blood change
- b Observe all vomitus and stools for gross or occult blood
- c Type and crossmatch the patient's blood carefully as soon as possible. Have whole blood or plasma available without delay
- d Obtain initial blood count and hematocrit initially and serially as indicated
- e Obtain blood N P N or urea nitrogen for comparison with later studies

#### ■ General Management

##### 1. Combat dehydration and salt depletion

- a Hypodermoclysis. Physiologic saline solution 1000 to 1500 ml daily by this method
- b Oral liquid feedings as soon as tolerated (see below)
- c Sodium chloride. 3 to 6 Gm ( $3\frac{3}{4}$  to  $1\frac{1}{2}$  dr) may be added to each liter of liquid food mixture to prevent salt depletion

##### 2. Nutrition

- a Starvation. The policy of initial starvation is subject to considerable controversy. Since the patient is often nauseated and anorexic or even in shock on the first day food may be safely withheld
- b Fluid. If patient is nauseated or vomiting thirst may be controlled by fluids given parenterally. The patient may be permitted to dissolve 1 teaspoon of hard fruit flavordandy under the tongue to relieve thirst
- c Diet. If the patient is hungry and not vomiting it is wise to begin immediate administration of bland foodstuff

(1) Liquid diet. It is best to begin with a liquid diet of hourly feedings of milk and cream mixture (see page 35), using supplementary antacid powders. Three to 6 Gm ( $3\frac{3}{4}$  to  $1\frac{1}{2}$  dr) of sodium chloride may be added to each quart of milk cream mixture to prevent salt depletion

##### (2) Solid bland foods

(a) Conservative approach. Solid bland food may be added when the patient has shown apparent clinical improvement on the liquid (milk and cream) regimen within 1 to 2 weeks and when the patient's stools have shown no occult blood for 3 to 3 days

(b) Liberal approach (e.g. Mullergraft). This method permits immediate feeding of all or intermittent high-calorie foods but in purified form

#### ■ Conservative Care. Following the acute episode the conservative medical regimen, such as outlined for uncomplicated peptic ulcer (see page 363) should be instituted.

#### D. Surgery. Surgery should be considered if

- 1 The general condition of the patient fails to improve despite the above measures
- 2 Bleeding persists as evidenced by gross or occult blood in stool. If the patient's condition permits gastrointestinal x-rays should be performed to help localize the source of identify the character of the bleeding lesion. Manipulation during such examinations should be as gentle as possible. If

## 270 Pyloric Obstruction

esophageal varices are eliminated as a cause of bleeding and the bleeding persists for more than 2-3 weeks prepare the patient promptly for surgical intervention. Do not wait until the patient becomes a poor operative risk before making this decision.

### PYLORIC OBSTRUCTION (code No 618)

It is important to differentiate pyloric obstruction due to spasm and edema from that due to scarring. The former condition may respond to medical treatment whereas the obstruction due to scarring is a surgical problem.

#### Treatment

##### A Medical Management (for obstruction due to spasm or edema)

1. Bed rest preferably in the hospital
2. Liberal use of antispasmodics
  - a. Oral. If patient is able to retain oral medication
    - (1) Tincture of belladonna 10-20 drops t.i.d. or q.i.d.
    - or (2) Belladonna extract 15 mg ( $\frac{1}{4}$  gr) t.i.d. or q.i.d.
  - b. Parenteral. If the patient is unable to retain medication by mouth atropine sulfate 0.3-0.6 mg ( $\frac{1}{200}$  -  $\frac{1}{100}$  gr) t.i.d. or q.i.d. subcutaneously

##### 3. Sedatives

- a. Phenobarbital (phenobarbitone) 15-30 mg ( $\frac{1}{4}$  -  $\frac{1}{2}$  gr) t.i.d. q.i.d.

- or b. Phenobarbital sodium (phenobarbitone sodium) 0.065 Gm (1 gr) subcut. every 8-12 hours p.r.n.

##### 4. Nutrition

- a. Sippy I diet should be used initially gradually progressing to Sippy II III and IV as tolerated (see page 53)
- b. Fluid or mineral imbalance must be corrected if vomiting is severe or prolonged. Parenteral methods are most satisfactory (see pages 10 and 27)
- c. Hypoproteinemia must be corrected since the resultant edema may increase pylorospasm.

##### 5. Control of hyperacidity

- a. Gastric secretions should be aspirated every morning and night with a small gastric tube. Some clinicians feel that continuous gastric suction should be employed initially.
- b. Antacids may be employed as for treatment of uncomplicated ulcer (see page 264) but avoid alkalosis from excessive use of soluble antacids since this increases pylorospasm.

##### B Surgical Measures (for obstruction due to scarring)

1. Surgery is to be employed only when a thorough trial of conservative measures has failed.
2. The various recommended surgical procedures will not be discussed. It is currently the practice to perform gastric resection in most cases although some surgeons favor gastroenterostomy.

# PERFORATION DUE TO ULCER

(Stomach

code No 640 951 3) (Duodenum

code No 651 951 3)

A cute h cut and h omi perfo tion of peptic ulc s may cc Acute pe fo ti co stit t s a medic i m g cy imme dial u gical repair p fe ably by simple s gi al clo ure is in d ated Mo e t nsi op rat ns re gene ally unwise at the time of the acut pis d b aus of the in r ed ope ati hazard due t the pat nts usually poo phys c i condition If th patient has b n r c living ACTH or tis n the py th d ugs must be imm diat ly dis ontin d If th p i t has h d no pr vious th r py o if p i u th apy has be n nadequat he may then be plac d upon a con ti m di l gm If th p ti nt has had an ad quat t ial of th apy p lor to the p isod pr p him fo pos ibl furth r t n i p ati p oced r s by t fusions and oth s pp rtie me su es Th t atment of s ba ut or hronic pe f ation may be m dic l u gical d pend g upo th pre nc o b ne of m pl cat o s ( g ab es i vol m nt f neighbo ing via e ) o pon th p t n nd s ty f ympt m

## GASTRITIS (code No 640 3 )

Th stomah m y be m ut ly h on ally fl m d du to a wide vari ty of pe fli and on pe flic Th sympt m of g t itis a poo ly d fin d nd i bl it i ot n al to h an ab n of ympt m wh n p s t th y lo ty mbl the ympt m of pepti ul

Physi al ami ation u ally id ag t X r y f nding a e not ema kabl unl s th g t t m pl i ted by at ophi hypert ophi c i l g h on h ng

Ga t i id ty m y b d a d i th h o i f m of g triti and blood m y b ob d in th g t ont is and in th f c s G i os opl i itation m y al ha t t m total hang wh h i m th ba fo i i i i if atio of g t i l l P ti nt with pe ni i u mia who h at ophi ga t itis must be ob rved f tly and pe i di lly f ide of malignant d g n ation of polyp

T m t

A Q ral Measure

e enti lly unila t that m pl yed in pepti ul t us ful (s p s 264)

B Sre i l l

g t r a s g i to R mo o timin t pe f a ti and ( g t u f t on lcohol tob )

## GASTRIC MALIGNANCY

(Carcinoma of the Stomach)

(code No 640 3 )

C rcinoma of the toma n ould be pe i d If pati ts over 45 year of g who develop dyspep Th di a more ommonly in m than in wom n Le ton or u ing region of the great r urvatu and p i i malignant A high inde of wpt ion f l x y

## 272 Diaphragmatic Hernia

studies and gastric analysis afford the greatest opportunity for early diagnosis. Unfortunately by the time the disease is manifest the fastases usually have occurred and the lesion is no longer amenable to satisfactory surgical therapy.

### Treatment

- A Specific Treatment (corrective) Early and thorough gastric resection is essential if the patient is a good operative risk. Patients should be afforded the opportunity of corrective surgery regardless of the apparently advanced nature of a malignant lesion.
- B General Measures (palliative) To be considered only when corrective surgery is impossible.
  - 1 Simple shunting procedures (e.g.: gastroenterostomy) in the event of pyloric obstruction.
  - 2 Symptomatic and supportive treatment as indicated.
  - 3 Narcotics should be given in adequate doses to alleviate suffering (see page 37).

## DIAPHRAGMATIC HERNIA

(Congenital code No 275 037 9) (Traumatic code No 274-424)

Herniation of a portion of the abdominal viscera through a congenital or acquired defect (especially esophageal hiatus) of the diaphragm may be manifested by a wide variety of symptoms but classically by epigastric distress and dyspepsia noted especially on lying down after meals. Nausea, vomiting, small hemorrhage and angina like symptoms may occur. X ray demonstration of the hernia is usually necessary to confirm the diagnosis. Small esophageal hiatus hernias which are of questionable clinical significance are reported frequently (at least 10%) on routine x ray gastrointestinal series.

### Treatment

- A Treat as for functional dyspepsia (see page 260)
  - 1 Small frequent feedings of bland easily tolerated food.
  - 2 Antispasmodic sedative medication (see page 266).
  - 3 Antacid powders frequently provide relief from heartburn (see page 264).
- B Instructions to Patients
  - 1 Patient should be instructed to
    - a Avoid lying down immediately after eating.
    - b Avoid exercising vigorously after eating.
  - 2 Patient should be advised to sleep in the semi Fowler position or at least with upper part of body slightly elevated in an attempt to decrease acid regurgitation into the esophagus.

## DISEASES OF THE INTESTINES

### REGIONAL ILEITIS (code No 654 952)

An acute or chronic inflammation of the distal portion of the

small intestine characterized by ulceration and scarring and often associated with internal and external fistulas. The condition must be differentiated from other specific causes of enterocolitis (e.g. tube colostomy chronic bacillary and amebic dysenterias). The history often of long duration, is one of mild intermittent diarrhea and abdominal cramps relieved by bowel movement. The acute form may simulate appendicitis.

Physical finding may include tender masses in right or left lower quadrants, fistulous tracts and perirectal abscesses. Occult blood is often present in the watery stools. Gastrointestinal x-ray series (small bowel study) reveals a loss of the mucosal pattern with narrowing and irregularity of the terminal ileum (string sign).

#### Treatment

A. Corrective. Radical primary resection of the involved portion of the bowel is the procedure of choice after a reasonable period of conservative medical therapy has been tried. Despite extensive surgical treatment the disease will recur fairly often.

#### B. Palliative

- 1 Diet. Bland, high caloric high vitamin adequate in protein.
- 2 Symptomatic treatment of anemia, diarrhea, avitaminosis as indicated.
- 3 Sulfonamides and antibiotics. Although of doubtful value those sulfonamides which are poorly absorbed from the gastrointestinal tract might be given a trial (e.g. p. 499). The effectiveness of streptomycin and Aureomycin® have not been completely evaluated.
- 4 Corticotropin (ACTH) and cortisone may produce beneficial results in certain patients with regional enteritis but results have been quite variable and generally not too encouraging. Experience would indicate that long term use of these agents may not be without hazard and may result in increased destruction of intestinal tissues.
- 5 Palliative surgery. Short cutting operations may be necessary when involvement is extensive and complicated.

### DIVERTICULOSIS (code No 660-642) DIVERTICULITIS (code No 660-643 0)

Multiple acquired or congenital evaginations (pouches) of the colon may occur at any place along the course of the bowel. Specifically the cecum. In diverticulosis the lesions are asymptomatic and are discovered accidentally on x-ray examination. Inflammation of diverticula (diverticulitis) with symptoms of intra-abdominal inflammation on referral to the involved site occurs mainly in individuals above 40 years of age. Variable lower gastrointestinal symptoms occur depending on location of diverticula. Abdominal pain and tenderness of the involved bowel disturbances require differentiation from acute appendicitis. Laboratory evidence of inflammation may be obtained and x-ray demonstration of diverticula helps to confirm the diagnosis.

except in those circumstances where there is a deficiency of these vitamins (due to inadequate food intake) It is felt that if vitamin B complex administration is indicated it is provided by the following

- (1) Dried brewer's yeast powder 30 Gm ( $\frac{1}{2}$  oz) daily or 30 0 8 Gm tablets daily
  - (2) Vitamin B complex high potency preparations
  - (3) Crude liver extract 1 2 cc I M 1 2 times weekly
  - (4) Choline and methionine as specific dietary supplements are of questionable value
- d Amino acid supplements (protein hydrolysates) may be incorporated in oral tube or parenteral feedings as indicated
- (1) Oral 2 15 Tbsp t i d (to supply 50 400 Gm daily)
  - (2) Tube 2 15 Tbsp t i d Rule out varices first
  - (3) I V 5% solution with 5% dextrose 1 3 liters daily
- e Skimmed milk may be used for oral or tube feedings
- f Salt poor albumin 10 100 Gm daily may be employed in severe cases (very expensive)
- g Ascitic fluid Readministration of ascitic fluid by sterile technic (if protein in ascitic fluid is greater than 1%)
- h Transfusions of whole blood if severe anemia and hypoproteinemia coexist
- 3 Ascites and edema may be treated by
- a Low sodium diet Reduce sodium intake to less than 2 Gm NaCl daily (see page 53) and even less if necessary even though diets severely restricted in sodium are apt to be nutritionally inadequate and unpalatable The danger of inducing the so called low salt syndrome with renal failure coma and death must be considered and watched for
  - b Attempt to restore plasma proteins to normal levels (as above) This is very difficult to achieve
  - c Cation exchange resins have a limited use when used, however the potassium resins should be employed since large doses of the ammonium resins may result in acidosis and even death Large doses of resins are necessary to achieve any significant sodium reduction
  - d Mercurial diuretics 1 2 cc I V or I M once or twice a week (see page 204)
  - e Abdominal paracentesis for pain discomfort or inability to eat if necessary
  - f Surgical procedures to relieve portal hypertension may be considered in selected good risk patients in younger patients in otherwise reasonably good physical condition in whom hepatocellular dysfunction is relatively slight the portacaval anastomosis may be employed with benefit
- 4 Anemia
- a Hypochromic anemia Ferrous sulfate 0 2 0 3 Gm ( $\frac{1}{2}$  gr) enteric coated tablets t i d p c
  - b Hyperchromic macrocytic anemia Crude liver extract 1 2 cc I M once or twice a week
- 5 Hemorrhagic tendency due to hypoproteinemia may be treated with vitamin K preparations although this treatment is ineffectual when intrahepatic damage is severe Blood

transfusions may be necessary to control the bleeding tendency. Some caution should be observed in using large doses of salicylate in the hepatic to be aware of the enhanced hypoprothrombin effect.

- a Oral Menadione U S P Menaphthone B P 13  
tablet of 10 mg ( $\frac{1}{10}$  gr) each t i d p c. In obstructive jaundice if present give supplementary bile salts (see page 286).
- b I V 1 M Menadion Sodium Bisulfite U S P 2  
mg ( $\frac{1}{100}$  gr) every other day
- 8 Hemorrhagic esophagial varices. Severe bleeding can at times be controlled by the use of the triple lumen tube. Surgical measures are usually hazardous and unsatisfactory.
- 9 Miscellaneous problems
  - a Pruritus (see page 87), nausea and vomiting (see page 231) and constipation (see page 254)
  - b Corticotropin (ACTH) and others. These agents should be used with full knowledge of hazards of hemorrhagic tendency, portal thrombosis and sodium retention. They are best not used in advanced cirrhosis.

### ACUTE CHOLECYSTITIS (code No 587.100)

Acute inflammation of the gallbladder may consist of any one of a wide variety of pathologic lesions of the gallbladder which are difficult to differentiate histically. The condition may develop as a result of obstruction of the biliary passage with or without some degree as a result of infection. Clinical findings may vary considerably in individual cases but for purposes of management it may be conveniently divided into 2 groups according to its mild, intermediate and severe.

#### Diagnosis

##### A. Symptom.

- 1 History of chronic dyspepsia or proved biliary calculi may or may not be elicited.
- 2 Attacks of right upper quadrant colic frequently nocturnal with residual gallbladder tenderness.
- 3 Nausea and vomiting are usually present during acute period.

##### B. Physical Examination

- 1 Tenderness during or following attacks.
- 2 Localized right upper quadrant tenderness is common.
- 3 Fever may be present or absent.

##### C. Laboratory Findings

- 1 Leukocytes in increased.
- 2 Erythrocytes elevated in common duct obstruction.
- 3 X-ray findings are variable and at times difficult to interpret. A gallbladder which fills poorly with the dye is suggestive of stenosis. Demonstration of stones in the gall cystogram is the most important finding.

#### Treatment

- A. Mild Type (Mild or indolent symptoms. Doubtful diagnosis)



- of acute colic
- 2 Abdominal distention
- 3 Nausea and vomiting
- 4 Intolerance of fatty and gas forming foods

#### B Laboratory Examination

- 1 Gallbladder dye demonstration of poorly functioning gall bladder (poor filling and emptying on repeated examination) and/or biliary calculi
- 2 Duodenal drainage may demonstrate excessive quantities of exfoliated epithelium mucus bacteria and pus in dark fraction of bile

### Treatment

#### A Medical Management

##### 1 Indications

- a Patients without clinical or x ray evidence of stones who respond to careful medical treatment
- b Questionable diagnosis or low grade symptoms Differ entiate from functional dyspepsia (a difficult problem)
- c Patients who refuse surgical treatment
- d Poor operative risk patients
- e Patients with a short life expectancy from other cause

##### 2 Treatment

- a Diet In general 2 different types of diet
  - (1) Low fat diet (classical type) This diet excludes both cooked and uncooked fats from all sources (see page 52)
  - (2) No-grease diet (modern concept) This diet excludes only the cooked fats (greases) which are non emulsified at body temperature but includes the uncooked fats such as are emulsified at body temperature The first phase of this diet is similar to the Sippy I diet with frequent feedings of milk and cream as improvement occurs the diet incorporates eggs butter cooked vegetables and fruit and cereals as tolerated
- b Antispasmodic medication Very useful
  - (1) Tincture of belladonna 10 drops t i d a c
  - (2) Belladonna extract 33 mg (1/4 gr) t i d a c
  - (3) Phenobarbital antispasmodic mixtures (see page 266)
  - (4) Atropine sulfate 0.4 to 0.6 mg (1/150 to 1/100 gr) orally sublingually or subcutaneously
- c Bile acid preparations Not to be used in patients with biliary stasis due to complete mechanical obstruction A cholagogue stimulates evacuation of the gallbladder a choleretic alters secretion of the bile constituents a hydrocholeretic alters volume of bile
  - (1) D hydrocholic acid N F (Decholin®) 0.33 Gm (3 3/4 gr) t i d p c choleretic (?) hydrocholeretic
  - (2) Ox Bile Extract U S P capsules 0.3 Gm (5 gr) or tablets 0.2 Gm (3 gr) t i d p c cholagogue choleretic and hydrocholeretic
- d Sedation Phenobarbital antispasmodic mixtures (see page 266) and barbiturates (see page 35)
- e Antacids These drugs frequently provide empiric relief of many of the annoying symptoms of gallbladder dyspepsia Their mode of action is not clear but they are felt to

relieve a gastric hyperacidity and to have a soothing effect on the duodenum and pharynx (see page 284)

f Five drugs (therapy)

(1) Sodium phosphate (disodium phosphate) 4-8 Gm (1-2 dr or 1-2 tsp) dissolved in warm water before breakfast

(2) Magnesium sulfate (Epsom salt) Dissolve 4-8 Gm (1-2 dr or 1-2 tsp) dissolved in warm water before breakfast This may be used initially but its prolonged use is inadvisable

g Local heat to abdomen Hot water bottle or electric pad preferred to mild anesthetic

### III Surgical Management

1 Indication (providing the patient is good surgical risk)

a Patient without clinical response to conservative treatment Surgical ultas however are questionable

b Patient with biliary stones with or without jaundice who recur with pain of right upper abdominal quadrant pain A symptomatic cholelithiasis in patients older than 45 years of age is considered by some to be an indication for surgery

c Patient with suspicion of gallbladder malignancy

2 Choice of operation is controversial. Generally, a laparotomy is preferred to the palliative procedures, but when the surgical risk is poor the patient is seriously ill, or there are clinical contraindications.

## DISEASES OF THE PANCREAS

### ACUTE PANCREATITIS (code No 580.830)

Acute pancreatitis is characterized by a sudden onset of severe agonizing constant epigastric pain often extending to the mid back shoulders or flanks. Symptoms of vasomotor collapse (shock) may be present. Paralytic ileus or obstipation and vomiting often occur. A pathologic picture of dyspepsia, ulcer, or gallbladder disease may be present. Physical examination reveals epigastric tenderness and rigidity. There is usually abdominal distention. There is an elevation of serum amylase and lipase levels (may be transitory). Leukocytosis is common and glycosuria may occur.

### Treatment

A Primary care is for dependent on shock (Vasomotor Collapse) (see page 31)

1 Bed rest in shock position (see page 3).

2 Morphine sulfate 15-20 mg (1/4-1/2 gr) subcut or if necessary 1 V may be employed for the relief of pain. Penthrone Demerol® 100-150 mg might be of value as a substitute for morphine sulfate because of its alleged antilepase modification.

3 Atropine sulfate 0.4-0.6 mg (1/250-1/100 gr) subcut should be given as an antispasmodic.

- 4 Glyceryl Trinitrate U S I (nitroglycerine) 0.3-0.6 mg ( $\frac{1}{100}$  -  $\frac{1}{100}$  gr) sublingually may be employed for relief of severe pain
- 5 Parenteral fluids
  - a Plasma Give 250-500 cc of plasma I V immediately and follow with subsequent infusions of plasma such as are necessary to correct disturbed fluid balance
  - b Crystalloids 5% glucose and/or normal saline may be used initially in lieu of plasma (when the latter is not available) or to correct altered fluid and mineral imbalance
- 6 Withhold food and fluids by mouth
- 7 Employ continuous gastric suction
- 8 Careful observation The patient should be constantly attended and vital signs should be checked at 15-30 minute intervals as indicated during the acute period. Blood count, hematocrit, serum amylase and lipase should be checked

**D Follow up** After the patient has recovered from shock or if patient has not developed shock 3 alternatives should be considered with regard to further immediate management

- 1 Conservative or expectant medical management This is to be preferred whenever possible. The patient should be observed closely for evidence of continued inflammation of the pancreas and/or related structures. The opinion of a surgical consultant should be obtained in every case of suspected acute pancreatitis
- 2 Immediate surgical intervention When the diagnosis is in doubt and there is a possibility of a serious and surgically correctible lesion (e.g. perforated peptic ulcer) an exploratory operation may be indicated
- 3 Observation The course of the inflammatory process should be observed by frequent repeated physical examinations and blood counts and by blood sugar levels and serum and urine enzyme determinations as indicated
- 4 Supportive therapy
  - a No fluid or foods should be given by mouth for at least 48 hours and continuous gastric suction should be maintained for that period
  - b After 48-72 hours small quantities of bland low fat liquid foods may be introduced gradually by mouth as tolerated. Gastric suction may be temporarily discontinued several times during the day for small oral feedings and then gradually discontinued depending upon clinical progress
  - c Fluid and electrolyte balance is maintained by appropriate parenteral fluids (see page 10)
  - d Atropine sulfate 0.4-0.8 mg ( $\frac{1}{150}$  -  $\frac{1}{100}$  gr) subcut may be administered t i d in an attempt to suppress pancreatic secretion

**C Convalescent Care** When clinical evidence of pancreatic inflammation has cleared

- 1 Bland low fat diet should be given
- 2 Drugs
  - a Belladonna extract 15 mg ( $\frac{1}{4}$  gr) t i d or atropine sulfate 0.4-0.8 mg ( $\frac{1}{150}$  -  $\frac{1}{100}$  gr) t i d
  - b Antacids may be of value (see page 264)

- 3 Evaluation of patient's surgery Consider the patient carefully for elective surgical treatment of biliary tract disease to help prevent recurrence of attacks

### Panophylaxia

- A All associated etiological factors should be corrected e.g. biliary tract disease duodenal ulcer etc
- B Diet Patient who has had previous mild attacks of acute pancreatitis should be placed on a low fat diet given and permitted no alcohol this may reduce the susceptibility of subsequent attacks

## CHRONIC PANCREATITIS (code No 580.956)

Chronic inflammation of the pancreas is associated with fibrosis of the gland. In the interlobular type the excretory secretions are often deficient and digestive disturbances are frequent. In the intralobular type the islets are involved and diabetes develops. Acute pancreatitis malignant disease pancreatic calculi penetrating peptic ulcer pepticobiliary disease and generalized arteriosclerosis are the more common causative factors. The syndrome is a history of recurrent episodes of epigastric pain and tenderness flatulence and bowel irregularities. The physiological examination is only an irregular intermittent titration.

Laboratory findings may include bulky foul fatty stools containing undigested food glycosuria and excess of pancreatic enzymes and duodenal drainage. Pancreatic calcification may be seen on x-ray.

### Treatment

A Specimen for Microscopic Examination None

B Diet Low Fat

1 Remove offending factors when possible

a Correct pepticobiliary disease

b Treat gastro-duodenal disease (e.g. penetrating peptic ulcer)

c Forbid the use of alcohol

2 Nutrition

a Diet: High CHO low fat, low protein, high calcium diet. When pancreatic achylia is the complication of failure of the islets protein hydrolyses may be employed to supplement natural protein. If diabetes is present dietary modification may be necessary (see page 13).

b Vitamins: Multivitamin tablets and B complex vitamins should be given.

c Calcium salts: Calcium gluconate 1 Gm (15 gr) tablets 2-3 tablets 3-4 times a day give to help replace calcium lost in stool.

d Replacement of deficient pancreatic enzymes: Pancreatin USP B.P. is available in finely powdered form, 0.32 Gm (5 gr) of the concentrate tablets 2-3 tablets 3-4 times a day. Dextrose is such a sorbitol monooxide may be used to correct the impaired fat and calcium absorption.



3 Drug

- a Ox Bil Est ct U S P (bil a lte) 0.5 Gm ( $7\frac{1}{2}$  gr) t i d p c may be of val
- b Hyd ochl ic Acid Diluted, U S P ■ P 1040 c (16-44 min) t i d with meals
- F r o u s l f e 0.203 Gm ( $3\frac{1}{2}$  gr) t i d p c for anemia
- d In lin for di b t s wh p sent ( ■ ■ 395)

PANCREATIC CARCINOMA (code No 690 ■ )

C rcinoma of th pancreas oc urs mo t ommonly in m l s ove 50 y rs of age. It is characteri d by epiga t i pain ext d ing to the ba k r pid and marked weight loss and m ltipl gastro int stinal complaints. Physiol mination m y rveal an plg s t ic mass let us and hep tic ■ rgement. Laboratory findings in l d vide ce of di tu bances of ca bhydrate m tabolism ele tion f s rum lipase and myl s and wid ning of duod nal loop o rre s configuration of duode um o ay

T m l

A Non ope tive M saur Symptom tic and palli tiv

B S g al M ur

1 R d l a g l scision in eet ct d c s s

2 Palliative surgi al ope tions Bill y t a t shunting p o-  
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## Chapter 11

# DISEASES OF THE URINARY SYSTEM

### NONSPECIFIC URINARY SYMPTOMS

Urinary symptoms should never be ignored. Symptomatic treatment must never be substituted for a thorough investigation of the underlying organic or functional abnormality.

#### FREQUENCY OF URINATION (code No 706) (Nocturnal code No 707)

Frequency is one of the most common of the urinary symptoms and may occur either during the day or night. It may be caused by any of a variety of organic or functional disorders and is often of psychogenic origin.

If the symptom is disturbing to the patient, treatment can be instituted while diagnostic procedures are being completed. Use antispasmodic sedative drugs as for dysuria (see below). Fluid restriction may be employed, particularly at night, if there are no contraindications.

#### DYSURIA (code No 704)

Dysuria may be caused by infection of the genitourinary system or by lesions of the lower urinary tract. It is usually associated with urgency and frequency. Mild discomfort may also be produced by a highly concentrated acid urine.

#### Treatment

A Specific Measures Treat the underlying disease.

B Symptomatic Measures Antispasmodic and sedative drugs

1 Atropine Sulfate U.S.P. B.P. 0.4 to 0.6 mg ( $\frac{1}{150}$  to  $\frac{1}{100}$  gr.) every 3 to 4 hours or other parasympatholytic drugs (see page 39)

2 Phenobarbital U.S.P. Phenobarbitone B.P. 15 to 30 mg ( $\frac{1}{4}$  to  $\frac{1}{2}$  gr.) t.i.d. q.i.d. or more as needed

3 Bladder sedative mixture

R Potassium citrate 30 g 51

Hyoscyamus tincture 30 0 51

Elixir of phenobarbital q.s. ad 120 0 5iv

Sig 4 cc (1 dr.) t.i.d. a.c. and h.s. or q. 4 H

## OLIGURIA (code No 702) and ANURIA (code No 703) (also see Lower Nephron Nephrosis page 303)

Oliguria and anuria are usually a late symptom and may be due to many causes. Shock, congestive failure and dehydration may all lead to anuria. It is important to differentiate these symptoms from urinary retention.

### Treatment

- A Sp ill Me a Tre t underlying disease
- B Fl d Do not give excess fluids to patients with oliguria or anuria due to renal failure. Death will result from overhydration. Simple dehydration with dextrose and saline solution (usually 5% dextrose in 0.9% saline) is effective. If necessary, dialysis may be employed. If the patient is in shock, any electrolyte loss (e.g. 31)

## RETENTION OF URINE (code No 705)

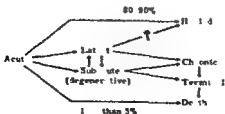
Urinary retention due to obstruction may be either partial or complete and is most commonly due to benign prostatic hypertrophy. The bladder may be palpable and the symptom is usually relieved by catheterization. If the procedure is contraindicated by other conditions, if the catheter cannot be employed, the patient should be treated by correction of underlying abnormality.

## SPECIFIC DISEASES OF THE KIDNEY

### GLOMERULONEPHRITIS

Glomerulonephritis is typically a diffuse glomerular disease involving both kidneys. The disease is believed by some to be due to a glomerular infection (usually bacterial) at the site of infection. The subsequent inflammation of the glomerulus is a progression of the uraemic disease.

The course of the disease can best be outlined by the following





It is difficult in the initial attack to predict the course of the disease. However, about 80% of the patients have mild damage which allows for healing. Most of the other 20% enter the so called latent phase and the nature of the progression of the disease seems to depend primarily upon the extent of the renal lesion. The greater the amount of initial damage, the more rapid the progression to subacute, terminal stages and death. By proper treatment it may be possible to retard this process.

### Diagnosis

Diagnosis of glomerulonephritis rests primarily upon the urinary findings of red blood cells and/or red blood cell casts; therefore, a careful examination of a freshly voided urine specimen is the best single examination in making the diagnosis.

### General Principles of Treatment

The problems of therapy in renal disease are threefold. Treatment of each aspect of the disease will be discussed in terms of these principles:

- A Addis's Principle of Rest. The cause of the progression of the renal lesion in glomerulonephritis is unknown. Dr. T. Addis suggested that progression is due to too great a work load for the amount of functioning renal tissue remaining. Most of the work of the kidney was assumed to be involved in the concentration of solutes (i.e., the reabsorption of water against osmotic pressure). Urea is the most important solute. Hence it was suggested that the less urea (from protein catabolism) and more water, the less work. However, it has recently been shown that the percentage of energy utilized by the kidney ( $O_2$  consumed) in the concentration of solutes is such a small fraction of the total as to make this thesis untenable. On the other hand, the empirical evidence of Dr. Addis's principles have not yet been refuted. Therefore, an adequate but minimal protein intake remains important in influencing the course of the disease.
- B Correction of Physiological Abnormalities. Since many of the manifestations of renal disease cause or are associated with marked physiological alterations (e.g., hypoalbuminemia in the nephrotic syndrome), therapy is aimed at correcting these physiological disturbances as they occur. However, many of the defects, apparently corrected, quickly revert to the disease state as soon as therapy is stopped (e.g., return of hypoalbuminemia after I.V. albumin is stopped). This may make continued intensive therapy imperative if one wishes to prolong life, especially in terminal cases. However, do not use therapeutic means which appear to correct the physiological defect but which are in themselves unphysiological or tend to defeat Principle A above (e.g., high protein diet to correct hypoproteinemia).
- C Complications. Complications are treated as they arise and are discussed in the appropriate sections.

## ACUTE GLOMERULONEPHRITIS (code No 712 100)

Diagnosis

The history usually reveals an onset of hematuria, puffiness about the eyes, headache and occasionally malaise and fever occurring 7-10 days after an infection (usually sore throat). The urine usually has a low specific gravity except when oliguria is present in which case it may be elevated. The urine sediment is loaded with red cells and white cells (total = 115) but has only few casts. Red cell casts pathognomonic. Mild to moderate proteinuria is present. There may be increase in blood volume and appearance of edema. The serum blood urea nitrogen and creatinine may be elevated. Hypertension is usually present.

Treatment

A Specific Measures There is no specific treatment. Treatment with corticosteroids (ACTH) or cortisone has been generally quite unhelpful. A few reports have been published, but these are exceptions. One case must be excluded in the use of corticosteroids (ACTH) or cortisone in this disease if overdoing can lead to increased morbidity and precipitate acute cardiac failure. Sodium restriction should be rigidly observed (see page 53).

B General Measures There is little that can be done to alter the abnormal physiology. Increase in blood volume, elevated NPN. The tendency to disseminate spontaneously the condition improves.

1. If necessary for hospitalization. The average case of acute nephritis can be cared for in the home although hospitalization becomes necessary if complication develops.
2. Bed rest. The patient should remain in bed but may have bathroom privileges. The duration of the period of bed rest is difficult to anticipate but it appears that urinary activity indicates the cessation of the renal lesion. A good general rule is to keep the patient in bed until the sedimentation rate is normal, until the blood pressure and blood NPN have been normal for about 2 weeks or until the urinary findings show at more than 0.2 Gm protein/24 hours. Do not wait for red cells to disappear from urine. If patient is eating, the diet is not important. They may remain for 12-14 weeks. Return to activity should be gradual.

3. Diet

Initially light diet with protein restriction indicated. 0.2-0.3 Gm protein/Kg (0.1-0.15 Gm/lb) body weight for first 7-10 days. Then increase to 0.5 Gm/Kg (0.15 Gm/lb) body weight plus the amount of protein lost in urine.

- a. Fluid and parenteral infusions (See page 112 for complete discussion).

- (1) Do not force fluids but allow fluids ad lib. Adjust fluid intake to changing physiological state (e.g. if oliguria develops fluid restriction may be necessary to avoid drowning the patient). In the acute phase diuretics cannot be given because of forcing fluid.
- (2) IV fluids are rarely indicated as a practical case.

associated with dehydration or abnormal fluid losses. In these cases it is important to give parenteral serum albumin 0.2 Gm /Kg (0.1 Gm /lb ) of body weight and sufficient glucose to maintain as close to an iso caloric intake as possible. Give I V fluids slowly.

- 4 Blood If anemia is marked a carefully cross matched blood transfusion may be administered slowly.

#### C Treatment of Complications

- 1 Cerebral edema with resultant headaches and convulsions
  - a If headaches are not severe and convulsions are not present give sedation with pentobarbital 15-50 mg (1/4-1 gr) or paraldehyde 4-8 cc (1-2 dr) i d to q i d as necessary.
  - b If headaches are severe and convulsions are present give magnesium sulfate 1.0 Gm (15 gr) (10 cc of 10% solution) I V slowly. **CAUTION:** whenever administering magnesium sulfate I V always have syringes filled with 10 cc of 10% solution of calcium gluconate or other calcium salt ready to administer I V if narcosis develops or respiration ceases.
- 2 Cardiac failure One of the most common causes of death in acute nephritis is acute cardiac failure. At the first sign of failure digitalization should be instituted by one of the rapid methods (see page 197). Sodium restriction should also be instituted immediately.
- 3 Focal infections Any focal infection especially of an acute nature should be treated promptly. Chemotherapeutic agents and antibiotics may be used as necessary for this purpose but they are of no value in the therapy of the nephritis itself. Effective blood levels of sulfonamides and penicillin may be maintained with smaller dosages than under ordinary clinical circumstances. This is due to the decreased tubular excretion (especially of penicillin) or the decreased glomerular filtration (as with sulfonamides).

### SUBACUTE GLOMERULONEPHRITIS (code No 712 100 0) (Nephrotic Syndrome code No 712 x40 or Degenerative Nephritis)

#### Diagnosis

A history of previous acute nephritis may not be elicited. The question of whether all cases of the nephrotic syndrome are preceded by acute nephritis has not been settled but it appears that they are not. The most common physical finding is marked pitting edema. Proteinuria is marked; the urinary sediment contains many casts (especially fatty casts) and many epithelial cells but few red cells. The blood N P N and creatinine are normal but the serum albumin is low and there is a marked lipemia. An anemia may also be found. Azotemia is also present in some cases.

#### Treatment

- A Specific Measures Corticotropin (ACTH) and cortisone have been employed in the treatment of subacute nephritis with

11. **Results** The use of these hormones results in a marked decrease or complete disappearance of the albuminuria with a subsequent or concomitant diuresis and a gradual return of serum albumin to normal level. The dosage and duration of administration are quite variable. The following are two main points of view:

1. **Intermittent therapy** The drugs are given in short courses of 7-14 days and repeated as necessary. Over 50% of patients so treated have relapsed promptly (see page 423 for dosage).
2. **Continuous therapy** The advocates of ACTH and cortisone therapy point out that this is a chronic disease and that there is no evidence that the drug in any way influences its ultimate course. The drugs are given continuously in the lowest effective dosage possible to keep the disease under control (i.e. urine free of protein).

All precautions for corticotropin (ACTH) and corticosteroid administration should be observed.

## B. General Management

1. **Fluids** The principle of rest for the kidneys is continued in this regard (see page 294). This stage may represent a temporary arrest of the renal lesion and lead into the terminal stage. The transition may be rapid or there may be recurrences of edema over a period of many years.
2. **Diet** Because of the massive albuminuria an attempt is made to keep the body in nitrogen balance but at the same time not to overload the kidneys. There is no evidence that the hypoproteinemia can be corrected by a high-protein diet per se. During long-term corticotropin (ACTH) or cortisone administration great care should be taken that the protein intake is adequate—**at least 1 Gm./Kg. (0.5 Gm./lb.)/day**.  
 Adult: 0.5 Gm. protein/Kg. (0.25 Gm./lb.) body weight per 24 hours plus an amount of protein per 24 hours that is equal to that lost in the urine. (Example: 70 Kg. man with proteinuria of 10 Gm./day = 0.5 Gm./Kg. body weight = 35 Gm. protein in 10 Gm. lost in urine = 10 Gm. protein. Total = 45 Gm. of protein per day as the approximate intake.)  
 Adolescent: 0.75 Gm. protein/Kg. (0.35 Gm./lb.) body weight per 24 hours plus urine loss.
3. **Treatment of edema** The prime aim of the physician is a physiologic point of view is the unimpeded removal of fluid from the intercellular and vascular spaces.  
 a. **Sodium restriction** This is probably the most important factor although not always effective when used alone. The sodium intake is restricted to below 1 Gm. (15 g.) per day 3 times a day or restriction to 0.5 Gm. (7½ gr.) per day may be necessary. This restriction should not be maintained longer than is clinically necessary and when the restriction is ended the patient should be watched for symptoms of sodium deficiency. Salt deficiency should not be overlooked. It is possible to lose 10 g. of salt in 1 day. In long-term cases exchangeable sodium present in the intercellular spaces is depleted and this is a procedure to be avoided.

- **Mechanical removal** Whenever the fluid accumulation becomes very marked mechanical removal is one of the most beneficial methods. This includes removal from pleural and peritoneal cavity and especially the use of Southey's tubes to remove massive edema from the legs. Any infection resulting from use of Southey's tubes should be controlled with antibiotic agents.

- **Agents to increase osmotic pressure of blood**

- (1) **Salt poor human albumin** Of all the measures that have been employed to increase osmotic pressure this agent has the soundest physiological justification. 30 Gm (1 2/3 oz) per day I V may induce a rapid diuresis. However the effect is transient most of the albumin is lost in the urine and much of the remainder is rapidly catabolized. After cessation of the therapy (may be continued for from several days or weeks) there is little evidence that the course of the disease has been modified and in most cases the serum albumin concentration will return to its former low level.
- (2) **Blood plasma** Plasma has little value primarily because of its high salt content. It may also carry the virus of infectious hepatitis.
- (3) **Some of the newer plasma expanders** (Dextran® Gelatin Plasmoid® etc) have been employed. Although they may induce a temporary diuresis their routine use is not yet indicated.
- (4) **Other preparations** such as acacia and isinglass are mentioned merely to be condemned. Their use is entirely unphysiological.

4. **Acid salts** (i.e. ammonium chloride) The  $\alpha$  drugs may be used but their effect is often not noted until mild acidosis develops. Since these patients may readily develop acidosis caution should be exercised in use of these drugs.

5. **Urea** Although urea is a diuretic it should not be used for it is the very agent that is being excluded when a restricted protein diet is given.

6. **Mercurial diuretics** are not advised. They may cause at least temporary renal damage and generally are not beneficial.

7. **Water** Patients should be encouraged to drink adequate fluids. As long as there is sodium restriction water will not accumulate in the tissues. Forcing fluids however is of little value in inducing a greater diuresis if fluid intake and urinary output are adequate.

8. **Induction of infection** It has long been known that patients with the nephrotic syndrome develop remissions following some virus infections especially measles. In susceptible children with subacute nephritis exposure to one of the mild exanthematous diseases may be indicated.

- **Treatment of Complications** The principal complications are infections commonly pneumonia and pneumococcal peritonitis. These should be treated with the appropriate chemotherapeutic and antibiotic drugs.

## LATENT GLOMERULONEPHRITIS (code No 712 190)

The patient with latent nephritis may or may not give history of an attack of acute glomerulonephritis. In the latent phase although there are no complaints or physical findings the lesion either has not healed yet or there is insufficient healing to carry the entire load of work. The latent phase may last for as long as 20 to 30 years and the patient may die of other intercurrent diseases before his renal function fails.

### Diagnosis

The only positive findings are occasional red blood cells and casts and a small amount of protein albumin. The physical examination and all blood findings (hematological and chemical) are normal.

### Treatment

#### A General Measures

- 1 Diet The patient should be on a minimal but adequate diet containing 55 to 75 Gm protein in/Kg (0.11 to 0.35 Gm/lb) body weight. At least 50% of the protein should consist of dairy products, vegetables and cereals.
- 2 Fluid The patient should be kept free of fluid. 3000 to 4000 cc (3 to 4 qt) per day.
- 3 Activity The patient should be cautioned against strenuous exercise but should be encouraged to live as normal a life as possible.
- 4 Physiological consolidation Since there is no positive physical laboratory support for the tentative diagnosis.

**B Complications.** Pseudo-complication of symptoms. Patients with latent nephritis have a characteristic response to any infection; this is particularly marked in the case of foreign protein reactions (e.g. vaccination inoculation, or infections). This reaction is characterized by hematuria (often gross) and a mild increase in proteinuria and white blood cells coming on immediately with the fever and subsiding with the fever. This is not another attack of acute nephritis. There is no delay between infection and renal reaction so hypertension seldom develops.

Most patients who have a second attack of true or latent glomerulonephritis. Most cases of so-called second attacks are really recurrences of late nephritis. These recurrences never damage the kidney eventually the initial attack and one can rarely detect any change in renal status after the attack is over.

- 1 Prophylaxis Because of the association of infection with fever infection and excretion, one should avoid the infection whenever possible. Patient with latent nephritis should not undergo vaccination routinely.
- 2 Treatment The treatment of the recurrences other than continued treatment of the latent nephritis. Treatment is aimed entirely at the precipitating cause. The patient should be kept in bed for about 1 week after fever has disappeared and should be allowed up slowly over the next week.

## CHRONIC OR TERMINAL GLOMERULONEPHRITIS

(code No 712 100 0)

It is difficult to say when the terminal or chronic stage begins. It is the time at which signs and symptoms of renal insufficiency develop. It may be very difficult to detect early, but as it develops certain findings appear. Most characteristic are the (1) elevation of blood N P N, (2) development of anemia, (3) gradual elevation of blood pressure, and (4) presence of a few casts and red blood cells in the urine. However, the blood protein is normal, edema is absent early, and there is slight proteinuria. This stage may last from several months to a few years.

### Diagnosis

A history of acute or subacute nephritis may be elicited. The physical findings vary with the severity of the disease, but hypertension with its associated vascular changes is the most common finding. Edema usually appears and may be due to cardiac or renal failure. The urine has a low or fixed specific gravity. There is a mild to moderate proteinuria. The sediment contains a few red cells and broad casts (renal failure casts) and a few epithelial cells. As anemia develops, increased blood urea nitrogen, alterations in electrolyte balance, and a decrease in serum proteins occur.

### General Treatment

- A Diet. As the blood N P N and creatinine begin to rise, any increase in protein intake is followed by a marked rise in N P N. The patient's protein intake must be restricted to 0.5 Gm./Kg. (0.23 Gm./lb.) body weight, plus the urinary losses.
- B Fluids are forced to 3,000-4,000 cc. (3-4 qt.) per day.
- C Treatment of Physiological Abnormalities. As the N P N continues to rise and renal failure becomes more severe, there is a progressive tendency to acidosis and altered electrolyte balance. The kidneys become unable to form ammonia or conserve fixed base, and fixed base elements consequently begin to decrease in the blood.
  - 1 Early in the terminal phase these are replaced by oral use of salts. Either of the following may be used:
    - a Calcium lactate 3.5 Gm. (45-75 gr.) daily
    - b A mixture of the following salts:
 

Sodium citrate	100.0 3xxv
Calcium chloride	3.0 gr. vi

 Sig: 2 Gm. (30 gr. or 1/2 tsp.) in 1 glass water t.i.d.
  - 2 Alkalinizing urine. The urine should be maintained at a pH greater than 8.0 with sodium citrate or sodium bicarbonate 1.2 Gm. (15-30 gr.) q.i.d. This is done to help prevent cast formation in the collecting tubules.
  - 3 Hospital treatment. As uremia becomes more marked and acidosis more profound, nausea and vomiting develop. It is generally necessary to place the patient in the hospital in order that the electrolyte balance may be adjusted as needed with I.V. fluids (see page 23). Uremia must also be treated (see next page).

## HEALED GLOMERULONEPHRITIS

Any patient who has had an attack of acute glomerulonephritis has undoubtedly suffered permanent destruction of some of the nephrons. The lesion is said to be healed when there is no longer any evidence of activity and the number of remaining functioning nephrons is great enough so that no impairment in function of structure can be found. However, the reliability of estimating how many nephrons this may be. It is always possible that the number functioning is barely sufficient to satisfy the everyday demands of the body.

### Follow-up

Subsequent to the attack may arise sufficient additional nephron damage to bring about a relapse of glomerulonephritis. Patients with healed glomerulonephritis must therefore be submitted to a moderate but not necessarily rigid protein restriction and should have urine examinations at least once a year for life.

## UREMIA (code No. 351)

Uremia is a physiological state resulting from renal insufficiency which may be defined as an imbalance in electrolyte balance with retention of nitrogenous and other waste products. Although uremia is most frequently seen in the terminal phase of chronic renal disease it does not necessarily imply an end stage of disease. Some cases of uremia may actually be due to a reversible condition resulting from a retention secondary to obstruction. In the management of uremia one should remember that the alterations in electrolyte balance are more important than the elevation of the BUN and that the therapy should be aimed primarily at preventing and relieving the clinical manifestations which develop.

### Pathology: Physiology of Renal Insufficiency

#### A Renal Insufficiency

1. Glomerular filtration is depressed and produces an elevation of the serum BUN, phosphorus, urea, and other nitrogenous wastes. This is due to metabolic acidosis (see page 30). The serum phosphorus rises and the serum calcium tends to fall.
2. Tubular function is depressed and the kidney loses its power to manufacture  $\text{NH}_4^+$  (which combines with fixed base) thus leading to loss of the fixed bases sodium, potassium, and calcium, which in turn contributes to acidosis (see page 30).

**B General Metabolic Effects:** Anemia develops gradually due primarily to bone marrow depression. Loss of calcium in urine with consequent low renal clearance and high serum phosphorus leads to parathyroid hyperplasia. Phosphorus cannot be excreted however and remains elevated. The serum protein becomes lowered.

#### Differential

1. Endopharmacology due to the clinical manifestation of uremia. There is a variable clinical picture which may be lethargy



heads be pruritus and weakness. Late uremia is characterized by acidosis and dehydration. In addition tetany may result from lowered serum calcium and muscular weakness may occur if serum potassium is lowered. The blood N F N sulfate and phosphorus are elevated the serum potassium is variable and the serum sodium calcium and  $\text{CO}_2$  are lowered. A normocytic anemia is present. Coma is superimposed later.

### Treatment

#### A Early

- 1 Diet Protein must be restricted to 0.5 Gm /kg (0.23 Gm /lb ) body weight plus the amount lost in urine. This tends to reduce BUN and serum sulfate (see page 294)
- 2 Fluids and electrolytes Force fluids orally to 3 000-4 000 cc (3-4 qt ) per day. Give calcium lactate and salt mixture by mouth as for terminal glomerulonephritis (see page 300). This helps keep the electrolytes in balance.

#### B Late

##### 1 General measures

- a Diet As above Protein restriction is very important
- b Fluids

- (1) Force fluids orally to 3 000-4 000 cc (3-4 qt ) daily unless patient is anuric
- (2) I V fluids and salts should be given as necessary to maintain normal electrolyte balance (see page 31)

##### c Electrolytes

- (1) Continue use of salt mixture (see page 300)
- (2) Aluminum hydroxide gel 15 cc (4 dr or 1 Tbsp ) q i d orally aids in reducing the hyperphosphatemia (causes precipitation of insoluble phosphates in bowel) and so helps to elevate serum calcium and prevent tetany
- (3) Calcium gluconate or lactate 10 cc (2½ dr ) of 10% solution I V is useful p r n to prevent tetany

- d Transfusions of carefully matched blood may be used to control anemia. All other forms of treatment to combat anemia are without benefit.

- 2 Complications of treatment In the treatment of uremia the physician is apt to encounter a therapeutic dilemma. In the course of attempting to correct the electrolyte balance the amount of sodium that must be administered may cause the patient to develop cardiac failure. Little can be done for the patient at this time he has almost no cardiac or renal reserve remaining.

#### C Termin 1

- 1 Calcium lactate or gluconate 10 cc (2½ dr ) of 10% solution I V p r n to control tetany and convulsions
- 2 Magnesium sulfate 1 Gm (15 gr ) (10 cc of a 10% solution) I V p r n for restlessness and convulsions. Caution: Have I V calcium salts ready in syringe (see page 296)
- 3 Paraldehyde 10 cc (5 dr ) in 30 cc (1 oz ) of oil rectally or 4-8 cc (1-2 dr ) I M as necessary for sedation

## EXTRARENAL AZOTEMIA

Extrarenal azotemia is the abnormal accumulation of nitrogenous waste products in the presence of normal or potentially normal renal function. The most common cause is a decreased effective circulating blood volume with inadequate glomerular filtration such as occurs in shock, dehydration, etc. The condition also occurs in massive gastric/intestinal bleeding where there is a sudden excess of protein digestion and absorption plus decreased circulating blood volume.

### Treatment:

Treatment is aimed entirely at the correction of the underlying condition rather than renal dialysis. Fluids and electrolytes sufficient to restore the blood chemistry to normal should be given.

## ACUTE RENAL FAILURE

(Lower Nephron Nephrosis code No 713 y00 2)

(Due to Hemoglobinemia Following Transfusion code No 713 38x 9)

### Pathological Physiology

It has long been demonstrated that the acute renal failure (liguriosis) which occurs in a variety of toxic conditions presents the same histological and pathological picture irrespective of the etiology. This condition is most often induced by one of the following: (1) intravascular hemolytic reactions (e.g. transfusion reactions); (2) rushing infusions; (3) burns; (4) chemical toxicity of some types (e.g. carbon tetrachloride, sulfonamide, etc.); (5) toxemia of pregnancy; and (6) non-traumatic mesenteric hemia. Although the pathogenesis is variable, the histopathologic picture is the same and consists primarily of focal degeneration of the distal convoluted tubule with blood casts in the lumen of nephrons and collecting tubules.

In most mild to moderate cases the kidneys will often spontaneously in 1 to 14 days (if the patient can be kept alive that long). In more severe cases the renal shutdown may be permanent. The evidence suggests that if the patient survives recovery is complete and that complete healing of the kidneys may occur in a short time, 2 to 4 weeks.

### Differential

- A. Acute Shock. At the onset symptoms of shock may be the only findings. Hemoglobin may be found in urine.
- B. Acute Renal Shutdown (May Last 14 or More Days). The initial symptoms are but if the shutdown persists the manifestations of uremia will occur: weight gain, peripheral edema, and the rales or pulmonary edema may be found. If the patient becomes overhydrated due to over-treatment with fluids, the drowning is the most common cause of death.
- C. Acute Recovery. The disease which follows renal shutdown may be mild and self-limited and may lead to dehydration, muscular weakness (due to low serum potassium) and tetany (due to low serum calcium) may occur. The blood BUN

usually does not return to normal until 2-4 weeks after initial recovery of the kidney has occurred

### Treatment

#### A. Emergency

1 SHOCK Since many cases are associated with traumatic or burn injuries the renal ischemia associated with shock may play a role in the pathogenesis. Immediate and vigorous therapy aimed at overcoming the shock is important (see page 31).

2 Immediate alkalization of the urine in cases of transfusion reaction may help prevent the precipitation of acid heme compounds in the renal tubules. Give sodium bicarbonate 5-10 Gm (75-150 gr) orally at once. Check the urine pH every 1-2 hours and give sufficient sodium bicarbonate to keep the urine alkaline.

B Oliguric or Anuric Phase The management of the patient in this phase is very difficult and should be carried out only by trained personnel in a hospital able to determine chemically the entire electrolyte panel (See page 21).

1 Weigh patient accurately daily. Weight gain means fluid retention and this must be avoided.

2 Fluid restriction This is one of the foremost principles in therapy. In the past patients were often drowned to death in an effort to promote diuresis. Usually 800-1500 cc of fluid is the maximum allowed daily. If patient is not losing excess fluids (as by vomiting, diarrhea, or excess sweating with fever) the insensible water loss is the only fluid which must be replaced. This can be calculated as follows: 0.8 Gm or 0.8 cc water/Kg (4 cc or 1 dr /15 lb) body weight per hour or 15 cc water/Kg (7 cc /lb) per day. This may be taken orally. If vomiting occurs the fluid may be given I.V. as 10-15% or more concentrated glucose given slowly and carefully so as to avoid infiltration. The amount given must be estimated clinically but under no circumstances should the patient be allowed to gain weight (keep an accurate record of weight) for this probably represents fluid retention. If patient is vomiting, has diarrhea, or is sweating, give additional fluids to replace this loss.

3 Electrolytes The electrolyte pattern should be examined daily and every attempt made to keep the electrolyte values within normal ranges. Give electrolytes as needed orally or parenterally. Give calcium gluconate 10 cc (2 1/2 dr) 10% solution I.V. for convulsions.

4 Diet A high carbohydrate and high caloric diet without protein will prevent endogenous protein breakdown and slow down the accumulation of protein breakdown products (i.e. urea, organic acids, and potassium). In the absence of vomiting a simple way to supply fluid and food is as follows: Pass a small polyethylene plastic tube intranasally into the stomach. Calculate the amount of fluid necessary over 24 hours and to this add lactose and salad oil to give the number of calories required for maintenance. This mixture may then be emulsified in a blender by adding 2-5 cc of Tween 80.

Urinary Tract Infections 305

5 Digitalization If any evidence of a di c failure cardia  
enlargement o pulmonary edema develop imm di te  
rapid digitalization and maintenance should be ried out  
(a e p g 197)  
Diuresis If diuretic has not been tried out  
it may be a consideration

It may be a necessary resort to maintain osmotic pressure to the 12-14th day of combat rising a of ml. Among those who have been advised the artificial kidney is the best. The use of oral exchange resin is final. And/or peritoneal irrigation is difficult to carry out but must at times be used if an artificial kidney is not available. The man

The man g m t f th diur tic  
A 24 hour u line sampl is llected  
The pool d in is then analyzed f total odium and pot alium  
content The amount of el ct olytes and w r r lost p r 24 hours  
is th n r plac d ver th n at 24 hou e This p ocedur i re  
peat d until renal f ctional i tivity has etu n d If th  
u in cannot be saved then the pati nt be id h e d fly s un  
As K and Cl d i rmin tions The fluid and lect lyt  
plac ment is d i min d on th basis of the m asu ments  
(s p g 21)  
Diu i m y oc ur in on of rene  
i Di i with in

The fluid and electrolyte balance of the body is maintained by the kidneys. The kidneys regulate the volume of fluid in the body and the concentration of electrolytes. The kidneys also regulate the acid-base balance of the body. The kidneys are the primary organs responsible for maintaining the fluid and electrolyte balance of the body.

The patient has a history of chronic renal failure and is on dialysis. The patient is currently on a low protein diet and has a serum creatinine level of 2.5 mg/dL. The patient has a history of hypertension and is on antihypertensive therapy. The patient has a history of diabetes mellitus and is on insulin therapy. The patient has a history of hyperkalemia and is on potassium-sparing diuretics. The patient has a history of hypocalcemia and is on vitamin D supplements. The patient has a history of anemia and is on erythropoietin therapy. The patient has a history of acidosis and is on bicarbonate therapy. The patient has a history of fluid overload and is on diuretics. The patient has a history of malnutrition and is on enteral nutrition. The patient has a history of depression and is on antidepressant therapy. The patient has a history of anxiety and is on anxiolytic therapy. The patient has a history of insomnia and is on hypnotic therapy. The patient has a history of constipation and is on laxative therapy. The patient has a history of dry mouth and is on saliva substitutes. The patient has a history of skin itching and is on antipruritic therapy. The patient has a history of hair loss and is on hair regrowth therapy. The patient has a history of weight loss and is on nutritional support. The patient has a history of muscle wasting and is on protein supplements. The patient has a history of bone pain and is on analgesic therapy. The patient has a history of fatigue and is on energy supplements. The patient has a history of weakness and is on strength training. The patient has a history of dizziness and is on vestibular therapy. The patient has a history of blurred vision and is on eye therapy. The patient has a history of hearing loss and is on hearing aids. The patient has a history of speech difficulties and is on speech therapy. The patient has a history of swallowing difficulties and is on swallowing therapy. The patient has a history of breathing difficulties and is on respiratory therapy. The patient has a history of heart failure and is on heart failure therapy. The patient has a history of liver failure and is on liver failure therapy. The patient has a history of kidney failure and is on kidney failure therapy. The patient has a history of endocrine failure and is on endocrine therapy. The patient has a history of immune system failure and is on immune system therapy. The patient has a history of nervous system failure and is on nervous system therapy. The patient has a history of reproductive system failure and is on reproductive system therapy. The patient has a history of sensory system failure and is on sensory system therapy. The patient has a history of motor system failure and is on motor system therapy. The patient has a history of integrative system failure and is on integrative system therapy. The patient has a history of all system failure and is on all system therapy.

**INFECTIOUS OF THE**

# INFECTIONS OF THE URINARY TRACT

## INFECTIONS OF THE URINARY TRACT

organisms may also cause infection. The diagnosis is usually suggested by the presenting symptoms and signs and is confirmed by the microscopic examination of the urine sediment and bacteriological examination of a sterile urine specimen.

A chronic or recurrent infection, particularly if resistant to antibacterial agents, suggests obstruction and urinary stasis. The final clearing of such infection is dependent upon the correction of the obstruction.

### General Principles of Treatment

- A Correction of structural abnormalities which produce stasis is of utmost importance in cases with remediable defects. Urinary tract infections may disappear spontaneously or be easily cured as soon as the defect is corrected. The permanent eradication of infection in the presence of such obstruction is usually impossible. The diagnosis of obstruction usually requires cystoscopy and/or excretion or retrograde pyelography. Treatment is generally surgical.
- B Treatment of the infection with suitable chemotherapeutic or antibiotic agents as determined by bacteriological studies.
  - 1 Careful examination of fresh sterile urine specimen (2nd glass specimen in male, catheterized specimen in female) for presence of pus and Gram's stain for preliminary identification of organism.
  - 2 Bacteriological identification of organism and determination of sensitivity of organism to antibiotic agents whenever possible. The latter is of special importance when streptomycin, Aureomycin® or Chloromycetin® are to be used, because adequate dosage must be assured to eradicate infection before organism resistance develops (see page 514).

## INFECTIONS OF THE KIDNEY

### Diagnosis

The manifestations of all infections of the kidney are similar but they vary in intensity with the severity of the infection. Symptoms include lumbar pain which usually radiates into the lower genitourinary tract but may radiate elsewhere, chills, fever, and nausea and vomiting as well as frequency, urgency, and dysuria. There is usually moderate to marked costovertebral angle tenderness. Examination of a sterile urine specimen for pus and organisms is necessary to make the diagnosis and to select the proper antibacterial agent.

- A Pyelitis (code No. 722.100) Simple infection of the renal pelvis which does not affect kidney function.
- B Pyelonephritis (code No. 719.100) Renal infection which depresses kidney function and which in the chronic form may produce effects similar to those of chronic glomerulonephritis.
- C Pyonephrosis (code No. 722.100.2) Renal infection of greater severity than pyelonephritis with pus in the renal pelvis. Renal and perirenal abscesses are surgical diseases of the kidney.

TreatmentA Specific Measures

- 1 Antibiotic therapy should be given as soon as causative organism is identified and as soon as sensitivity tests have been conducted to determine dosage (see page 314)
- 2 Surgical treatment of any removable obstruction should be carried out when acute symptoms have subsided. Diagnostic studies of the urinary tract should be deferred until the acute phase has passed

B General Measures

- 1 Bed rest until completely asymptomatic
- 2 Fluids If the kidney function is not depressed and there are no other contraindications fluids should be forced. Maintain daily urinary output of 1500 cc or more
- 3 Analgesic and sedative as necessary for the comfort of the patient

- C Treatment of Chronic Pyelonephritis Therapy consists of chronic pyelonephritis in which the kidney has been moderately to markedly damaged. Infection in the kidney is very difficult to eradicate. Additional suggested the continued use of small doses of a sulfonamide drug 100-200 mg (1½-3 gr) tid qid (if other measures have been taken to eradicate the infection) in the hope that the small doses might stop or slow down the progression of the disease. Once this therapy is begun it should probably be carried on for life.

Terminal pyelonephritis is handled the same as terminal glomerulonephritis (see page 300)

**CYSTITIS**

(Acute code No 730 100) (Chronic code No 730 100 0)

Definition

Inflammation of the bladder is many times more common in women than in men and is most commonly due to Escherichia coli. It must be differentiated from urethritis which has similar manifestations

- A Symptoms Mainly dysuria, urgency and frequency. If severe the patient may have at times urinary retention. Chills and fever may occur. When infection is very severe hematuria may develop
- B Signs Suprapubic tenderness may be present
- C Laboratory Examination
- 1 Organism and pus will be found in the urinary sediment of a properly collected specimen
  - 2 Organism must be identified by a combination of stained smears (methylene blue or Gram's) and by culture for purposes of isolation of the organism (see page 314)
  - 3 Two-glass test may be used to differentiate urethritis from cystitis in the male. Examine the urine grossly and microscopically. If the urine in the second glass is cloudy the infection is in the urethra. If the urine is turbid, the bladder is infected (U. S. color is dark red or glassy)
  - 4 First glass contains at least 4-6 cc of urine and contains the elements from the urethra

- Second glass contains the remainder of the urine from the bladder
- A third glass may be collected after prostatic massage. In this method the patient must retain some urine in the bladder to wash out any residual material
- Cystoscopy May be necessary to determine the presence of obstruction, upper urinary infection, or source of bleeding. This must not be done during the acute phase.

### Treatment

#### A Specific Measures

- 1 Antibacterial agents. Select the appropriate drug by bacteriological examination and sensitivity tests (see page 314)
- 2 Surgery. Correct any remediable obstruction after the acute stage has subsided.

#### B General Measures

- 1 Bed rest if severe
- 2 Fluids. If urination is painful, fluids should not be forced. When dysuria has subsided, maintain a high urine output.
- 3 Bladder sedatives and analgesics
  - a For severe pain. Mild local anesthesia can be obtained by bladder instillation of 2% solution of tetracaine Hydrochloride® or 1:1000 (0.1%) solution of Nupercaine®. Allow the anesthetic to remain in the bladder for 10 minutes by placing a clamp on the catheter. After draining off the anesthetic, instill 10 cc of 5% solution of mild silver protein or 1:10,000 silver nitrate solution and leave in the bladder.
  - b For tenesmus. Treat as for dysuria (see page 292)

## **TUBERCULOSIS OF THE URINARY TRACT**

(Kidney code No 710 123) (Bladder code No 730 123)

Chronic tuberculosis of the urinary tract usually occurs first in the kidney and involves the bladder secondarily. A history of bladder irritation is usually present; the urine contains pus and a few r b c, but there generally are no organisms. The urinary sediment must be examined microscopically and bacteriologically (culture and guinea pig inoculation) for acid fast bacilli. If tubercle bacilli are found, determine the primary urogenital site of the infection and whether renal disease is unilateral or bilateral.

### Treatment

- A Treatment of renal tuberculosis with the newer anti-tuberculous chemotherapeutic agents stops progression and may effect cures in some cases. Therapy is the same as that advocated for pulmonary or other systemic tuberculosis. The use of intermittent streptomycin plus para-aminosalicylic acid for long periods of time (1 to 3 years) has been used most extensively. 1 Gm (15 gr) streptomycin for 0.5 Gm streptomycin and 0.5 Gm dihydrostreptomycin every 3rd day and 12 Gm (360) PAS daily in divided doses seems to be an adequate schedule.
- 1 Surgery. If unilateral tuberculosis is found and if the kidney is seriously involved, nephrectomy with subsequent streptomycin

therapy should be considered. This would appear to help to cure any lower tract tuberculosis that may be present.

C Symptomatic and supportive measures as necessary.

## OTHER DISORDERS OF THE URINARY TRACT

### CARCINOMA OF PROSTATE

(Adenocarcinoma code No 764 8091)

#### Diagnosis

The vast majority of prostatic carcinomas can be diagnosed by the finding of a hard gland or area within the gland on rectal examination. Confirmation of the diagnosis is made by needle biopsy and the finding of an elevated acid serum phosphatase. X-rays of the pelvis and lower spine are taken to determine the presence of metastases.

#### Treatment

A Early Cases Treatment of choice is radical surgical removal. This is reserved only for cases in which (1) there is no evidence of metastases (2) the gland is not fixed to surrounding tissue (3) the patient is otherwise a good surgical risk and has a good life expectancy.

B All Others C

1 Hormonal therapy. The estrogens have been found to be of great benefit in relieving the pain associated with metastases and in arresting the progress of the disease (in some cases actually causing a regression). The widest experience has been gained with Diethylstilbestrol (U.S.P. Stilbestrol, B.P. and Ethinyl Estradiol, N.N.R.).

a Dosage

- (1) Average maintenance dosage: Diethylstilbestrol 1 mg (1/80 gr) or ethinyl estradiol 0.1 mg (1/200 gr) per day orally for life.
- (2) If no or poor response may increase as follows: Diethylstilbestrol 2-3 mg (1/30-1/20 gr) or ethinyl estradiol, 0.2-0.3 mg (1/300-1/200 gr) per day.
- (3) Some authorities feel that once the therapy has been given, diethylstilbestrol 5-10 mg (1/12-1/6 gr) or ethinyl estradiol, 0.5-1.0 mg (1/120-1/60 gr) per day.

b Toxic signs

- (1) Nausea and vomiting may occur but usually disappears as the drug is continued. If severe, diminish dosage and increase as tolerance develops.
- (2) Puffiness and enlarged breasts usually develop on continued dosage but this is no contraindication to continued therapy.

- 2 Orchiectomy. The results to date agree that bilateral orchiectomy plus estrogens is of great value than estrogen alone.
- 3 General supportive measures such as surgical removal of a prostatic urinary obstruction etc. should be employed.
- 4 Bilateral orchiectomy and adrenalectomy (with cortisone and estrogen maintenance) in order to remove all possible



androgens must still be considered experimental. To date the results in most cases seem little better than orchiectomy plus estrogens alone.

### UROLITHIASIS

(Renal Calculus code No 719-615) (Ureteral Calculus code No 723-615) (Renal Colic code No 711)

Renal colic is usually caused by the passage of a renal calculus into and down the ureter. It is characterized by a sudden onset of severe pain in the lumbar region of the affected side radiating to groin bladder testes inner thigh, or to other adjacent areas. The pain requires narcotics sometimes in large doses for relief. Nausea and vomiting may occur but no other constitutional symptoms are present unless there is a pre-existing infection. Urine output is reduced and hematuria is commonly seen. Some stones may pass without symptoms.

#### Treatment

##### A. ANALGESIC MEASURES

- 1 Narcotics for relief of pain. These may have to be repeated if pain is severe.
  - a Morphine sulfate or hydrochloride 15 mg ( $\frac{1}{4}$  gr) I V or subcut Stat. Atropine sulfate 0.5 to 0.75 mg ( $\frac{1}{80}$  to  $\frac{1}{40}$  gr) may be given with the morphine.
  - b Meperidine Hydrochloride Injection U.S.P. (Demerol® Dolantin®) 0.100 Gm ( $\frac{1}{2}$  gr) I M or orally in place of morphine. This has a minor atropine like effect in addition to its narcotic action.
- 2 Heat over the affected flank and low abdominal area may give some relief. This can be given as warm moist towels, heat pad or warm tub bath.

##### B. General Measures

- 1 Fluids. If patient does not develop anuria or oliguria fluids should be forced in order to maintain a high urine flow. Fluids should be given I V if vomiting prevents oral administration. Any individual who has had a renal calculus should be encouraged to drink large amounts of fluids at all times.
  - 2 Check carefully for passage of stone. If this does not occur patient should be examined by x ray for position of stone.
  - 3 Attempt to recover stone by having patient void through a funnel layered with several thicknesses of gauze.
- Surgery If a stone becomes lodged in the ureter it should be removed surgically to prevent hydronephrosis.
- D Coexisting infection should be treated with suitable antibacterial agents (see page 514).

#### Prophylaxis

- A Correction of Underlying Disease Treat any disorder which may cause or assist stone formation. These include hyperparathyroidism (see page 377) urinary obstruction and urinary infection (see page 306). Every patient with urinary tract calculi should have at least one serum calcium and phosphorus determination to rule out hyperparathyroidism.
- B Fluids Any patient who has had a renal calculus must be encouraged to drink large amounts of fluid at all times.

## Chapter 12

# DISEASES OF THE MUSCULOSKELETAL SYSTEM

## INTRODUCTION

### Classification of Rheumatic Diseases

- 1 Acute or chronic rheumatism
- 2 Arthritis due to rheumatic fever
- 3 Acute rheumatoid arthritis
- 4 Degenerative joint disease
- 5 Acute disseminated gonorrhea
- 6 Arthritis due to gout
- 7 Other arthropathies (due to drug, neoplastic metastasis, bacterial, hemolytic, allergic, toxic, and unknown cause)
- 8 Fibrositis, myositis, bursitis

### Examination of the Patient

The examination of the patient with rheumatic disease should include a careful history and physical examination with special emphasis on determining the functional status of the joints (e.g., of motion, x-ray, and deformity, atrophy, etc.). Routine laboratory tests include a blood sedimentation rate and x-ray of one or more of the involved joints. It is essential to complete the diagnostic picture. Additional studies may include determination of blood urea nitrogen, aspirin, and examination of joint fluid. Differential diagnosis is important not only from a diagnostic standpoint but also serves to provide a basis for planning the therapy and evaluating the clinical progress of the patient.

The differential diagnosis of the four major forms of arthritis are to be found in the table on page 312 and 313.

## RHEUMATOID ARTHRITIS (code No 24 1 0)

Rheumatoid arthritis (commonly known as rheumatism) is a chronic disabling systemic disease of undetermined origin. It is ordinarily considered as involving primarily the joints, but it is actually capable of involving most of the tissues of the body, particularly those of mesodermal origin, including lymph nodes, bone marrow, liver, pleura, gastrointestinal tract, endocrine system, myocardium, kidneys, connective tissue, and the musculature. The disease may involve any or all joints and is of varying severity. The characteristic feature is pain in the

DIAGNOSTIC CHARACTERISTICS OF THE MAJOR FEATURES OF ARTHRITIS

	Rheumatoid Arthritis	Arthritis Due to Specific Infection	Degenerative Arthritis	Arthritis Due to Gout
Family history of similar condition	+		+	+
Past history	Frequent infections	History of specific infection		
Sex	Most common in women	Either sex	Both sexes	Usually men
Age at onset	Any age usually 20-50	Any age	Usually over 40 y are	Usually over 35 year
General physical status	Poor undernourished	Acute good	Good but may show other a ntle change	Good
Type of onset	Insidious (subacute)	Acute infection sudden	Insidious (slow)	Sudden (cessation of symptoms also sudden)
Fever	+	Chronic infection slowly + (especially acute)		+ (during acute episodes)
Joints involved	Any joint often symmetrical with tendency to spread centripetally Proximal finger joints especially involved	Any joint pyogenic forms are usually monarticular Non pyogenic forms are often polyarticular	Usually the large and weight bearing joints Also distal joints of fingers	Any joint monarticular or polyarticular Especially involves metatarsophalangeal joint of great toe
Periarticular swelling	+	+		+
Ankylosis	+	+ (pyog nlc)		
Muscle atrophy	+	+		
Deformities	+	+ (pyog nlc)		+ (late)

Cutaneous changes		Disorders of integument and glands	Similar to rheumatism	Systemic changes	Early Non Lat Siml r to h m fold arthritis
Subcutaneous nodules					
Leucoderma					
Blood dyscrasias					
Hypertrophy of the heart					
Hypertrophy of the lungs					
Hypertrophy of the liver					
Hypertrophy of the spleen					
Hypertrophy of the kidneys					
Hypertrophy of the pancreas					
Hypertrophy of the thyroid gland					
Hypertrophy of the parathyroid glands					
Hypertrophy of the adrenal glands					
Hypertrophy of the pituitary gland					
Hypertrophy of the hypothalamus					
Hypertrophy of the brain					
Hypertrophy of the spinal cord					
Hypertrophy of the peripheral nerves					
Hypertrophy of the lymphatic system					
Hypertrophy of the endocrine system					
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## 314 Rheumatoid Arthritis

peripheral joints early in the disease and ankylosis and deformity are common end results

### Diagnostic Features

#### A Clinical Manifestations (See also the table on page 312-313)

- 1 Non-articular manifestations include weakness, anorexia, fever, weight loss, clamminess of skin, muscular aches and tremors, iritis, migratory pleurisy, lymphadenopathy, anemia, and involvement of any of the other above mentioned body tissues
- 2 The acute form of the disease is rare but may run a severe fulminating course associated with high fever, chills, cachexia, and a rapid death
- 3 Mild or transient types of rheumatoid arthritis may occur
- 4 Although certain joints are classically involved, any or all joints may be involved. Joint involvement may be monarticular, but this is rare
- 5 In rheumatoid arthritis of the spine (rheumatoid spondylitis) the patient may or may not be otherwise healthy but will develop recurrent low back pain associated with progressive stiffness of the spine and reduced chest expansion, often without significant involvement of the peripheral joints

#### B Laboratory Data

- 1 Increased blood sedimentation rate and, less commonly, leukocytosis are considered to be evidence of clinical activity
- 2 X-ray changes of joints and periarticular structures may be quite characteristic (see page 313) and helpful in differentiation from osteoarthritis, although osteoarthritic changes may occur coincidentally in rheumatoid arthritis and thereby confuse the picture

Narrowing of joint spaces and ankylosis of the sacro-iliac and apophyseal joints and calcification of the anterior and lateral spinal ligaments may be demonstrated in rheumatoid spondylitis

### Treatment

#### A General Measures

##### 1 Rest

- a Acute illness: Complete bed rest should be reserved for the patient with the acutely active or severe rheumatoid arthritis. Special care, including exercises, should be used to prevent deformities in bed patients, and the affected joints should be placed in the optimal functional position
- b Mild chronic illness: 1-2 hour rest periods during the daytime as well as 10-12 hours rest in bed at night are essential. Analgesics and sedative drugs (not narcotics) and physical therapy may be used judiciously to insure relaxation, rest, and sleep

- 2 Physical activity: Carefully regulate the daily schedule of activities of the patient and allocate periods for work, play, and exercise as well as for rest

- a Ambulatory patients: It is usually necessary to specify the hours and physical limitations for ambulatory patients according to the demands of the individual case

- Bed patients It is imperative to institute a program of daily systematic exercises to prevent joint stiffness and muscle atrophy. Refer to the section on physical management of arthritic joints (see page 323).
- 3 Diet Food should be simple, nourishing and palatable. An adequate protein and high vitamin diet is usually feasible. Stomach and intestinal disorders are frequent in rheumatoid arthritis. It is often necessary to modify the diet to tolerate food which would otherwise increase or decrease according to the patient's weight.
- 4 Dietary supplement  
a Iron salts may be indicated if anemia is present (see page 219).  
b Multivitamins The use of a acceptable multivitamin preparations as a general health building measure may be indicated although it is probable that no one of the vitamins has specific therapeutic effect on this condition.  
Vitamin D High potency vitamin D preparations in daily dosage ranging from 50,000-300,000 unit in divided doses. There is no popularization of being of great value. Toxicity of these vitamin D compounds in prolonged or excessive doses is definite and their effectiveness has been questioned by many investigators. Other individual vitamins have failed to demonstrate significant beneficial results.
- 5 Elimination of precipitating factors  
a Infection Evaluate the role of systemic or focal infections only as they may precipitate the individual patient. Eliminate potential infections whenever possible. Finally, infected tonsils etc. may be removed or irradiated. In addition, it is best to maintain a conservative attitude toward the elimination of questionable focal infections. Usually when their correction will involve extensive or major surgery. Anti-infective agents should be given only to combat specific infection and not the rheumatoid disease.  
b Psychogenic factors Frequently rheumatoid disease has its onset when the patient is working and living in a highly stressful environment where he is subjected to undue emotional stresses or tensions.  
c Improve living hygiene For correction of such factors see sections on diet, physical activity and diet (above).
- 6 Psychotherapy  
a Reassure patient and relieve existing anxiety.  
b Regulate patient's environment to minimize emotional distress.  
c Keep an optimistic and cheerful attitude.  
d Explain the nature of the disease and the role of the patient himself in overcoming his illness.  
e Enlist aid of a trained psychiatrist in appropriate cases.
- 7 Relief of pain Avoid narcotics  
a Analgesic drugs Give analgesics liberally if role is to relieve pain as an aid in preventing muscle spasms and deformity.  
(1) Sodium salicylate 2-6 Gm (10 gr) 4 times a day coated to prevent gastric distress every 2-4 hours for pain.

(2) Aspirin 0.3-0.6 Gm (5-10 gr) every 2-4 hours  
p r n pain

(3) Analgesic sedative mixture

R Sodium salicylate 10-15 3iiss iv

Elixir phenobarbital q s ad 120 3iv

Sig 4 cc (1 dr or 1 tsp) every 4 hours p r n

- b Sedative drugs Barbiturates can be used effectively in enhancing the action of the analgesic drugs Pheno-  
barbital 15-30 mg ( $\frac{1}{4}$ - $\frac{1}{2}$  gr) 3-4 times daily
- c Physical therapy Physical methods utilizing local heat  
to involved joints and proper splinting are effective in re-  
lieving pain and muscle spasm (see pages 323-334)
- d X-ray therapy is of no value in peripheral joint involve-  
ment of rheumatoid arthritis in rheumatoid spondylitis  
however deep or penetrating x-ray therapy carefully  
administered in repeated courses has proved to be of  
value This treatment must be administered only by  
trained x-ray therapists

**B Hormone Therapy** The hormonal and steroidal agents which  
are used in the treatment of rheumatoid arthritis although  
representing a significant advance must be considered as only  
ancillary measures to the comprehensive approach to the treat-  
ment of this disease Perhaps the greatest disadvantage which  
might stem from their use aside from the very serious prob-  
lem of untoward reactions lies in the tendency of patient and  
physician alike to neglect the less spectacular but proved bene-  
fits which may be derived from general support, i.e. treatment  
physical therapy and orthopedic and other measures These  
agents do not represent the long awaited specific anti-  
rheumatic factor and do not cure the disease

- 1 Cortisone acetate and hydrocortisone produce startling re-  
sults in acute rheumatoid arthritis but unfortunately when  
the drugs are discontinued the condition regresses quite  
promptly Subjective improvement may be experienced with-  
in 6-48 hours after the initial dose but objective changes  
such as increased mobility of joints and diminished joint  
swelling occur more slowly and less constantly The period  
of remission following discontinuation of these drugs may  
vary from a few days to a few months It would seem that  
the principal indications for hormonal therapy in rheumatoid  
arthritis are for the control of the acute exacerbations of the  
illness and the prevention of rapid progression due to exten-  
sive inflammatory reaction Optimum dosage schedules have  
still not been established although it would now appear that  
satisfactory results can be obtained with smaller or more  
conservative doses than those employed previously It is  
recommended that initial doses of 75-100 mg (orally or by  
injection) daily be used until adequate control is achieved  
Maintenance levels of 25-100 mg daily may be continued in  
some cases indefinitely Some observers feel that rest  
periods of 1 month between the 4-8 week courses are advis-  
able However the drugs have been employed continuously  
now in many patients for several years without apparent  
harmful effect (see pag 423 for further discussion of phys-  
iology dosage toxicity etc of cortisone)

- 2 Corticotrophin (ACTH) Striking results similar to those obtained with cortisone have been reported (F = recom mended dosage = pag 423)
- C Gold Therapy (Chrysotherapy) Although gold salts have been used extensively in rheumatoid arthritis their value remains highly controversial
- 1 Indication Gold therapy is indicated for active rheumatoid arthritis only. Some clinicians feel that it should not be used until a reasonable trial of conservative measures has failed to achieve the desired results. Others feel that the chances for complete remission may be better if gold therapy is used early in the disease.
  - 2 Contraindications Nephritis, hepatic insufficiency, blood dyscrasias (including anemia), hemorrhagic tendency, pregnancy, strong personal history of allergy or allrgies, severe diabetes mellitus, a general skin disorder, ulcerative colitis, cutaneous fever and tuberculosis.
  - 3 Preparations and dosage The gold salts are usually given on a week beginning with small doses. Indicated below. The weekly dose is increased gradually until the maximum optimum dose is being given. This amount is then continued weekly until the desired response is obtained. The maximum amount is given so that reactions occur. On one or two or more courses of 1000 to 2000 mg of gold salt give with a rest of 2-3 months between courses. The value of the maintenance dose is small. It is given at regular intervals. Undesired side effects are among the most serious of chrysotherapy although many workers are employing this plan at present.

## GOLD PREPARATIONS

Preparation	Route of Injection	Clinical One Dose Each Week		
		First Week Dosage	Amount of Increase per Week	Optimum Maximal Weekly Dose
Gold Sodium Thio-sulfate (37% gold in aqueous solution)	I M o I V	5 mg	5 mg / week	Females 30 mg Males 75 mg
Gold Sodium Thio-maleate (50% gold in aqueous solution)	I M only	10 mg	Increase to 25 mg in 2nd week if tolerated, increase to 30 mg in 3rd week	30 mg
Gold Sodium Thio-glucose (50% gold in oil suspension)				

- 6 Toxic reaction An average of 37% of patients (range in various series 8-61%) experience toxic reaction. The mortality rate is about 0-4%. The toxic effects of chrysotherapy include those of other heavy metals notably: rash (see page 335) and toxic dermatitis (rashes, bullae, leukoderma, agranulocytosis, purpura, hepatitis, etc.)



reactions bronchitis aplastic anemia peripheral neuritis nephritis and photosensitization

- a Reduction of frequency and severity of toxic reactions
  - (1) Observe for the contraindications mentioned above
  - (2) Observe patient carefully during the course of gold therapy and for a period of several weeks thereafter
    - (a) Complete medical examination prior to therapy
    - (b) Before each subsequent injection ask patient how he has felt since the previous injection examine the skin and mucous membranes for dermatitis or purpura and examine the urine for albumin and microscopic hematuria
    - (c) Every 2 weeks obtain Hgb WBC and differential
    - (d) When indicated perform special tests such as platelet counts or liver function tests
  - (3) Warn patient against exposure to strong light
  - (4) Withdraw drug immediately if any toxic reactions appear Wait for a few weeks if reaction is mild and clears promptly treatment may be resumed with small doses
  - (5) There is no known method of decreasing the tendency to toxicity in a given individual except perhaps through reduced dosage
- Treatment of toxic reactions
  - (1) Withdraw drug immediately if early toxic reactions appear
  - (2) Treat reactions as for arsenical poisoning (see page 536)
    - (a) Try BAL® on all cases (see page 536)
    - (b) For treatment of agranulocytosis see page 231
- c Masked toxicity If gold salts are used during hormonal therapy a toxic reaction may be masked appearing with explosive violence when the hormones are stopped Therefore use gold salts with great caution during hormonal therapy

### OSTEOARTHRITIS (code No 240 912)

A chronic degenerative joint disease of undetermined cause usually of late adult life associated with varying degrees of symptoms and/or disability of multiple joints Ankylosis of joints does not take place except in the spine

#### Diagnostic Features (See table on page 312-313)

- A The disease may exist with a complete absence of symptoms when symptoms are present they are usually mild
- B Joint Symptoms Included
  - 1 Stiffness which improves with mild activity
  - 2 Aching and pain aggravated by overactivity or injury and relieved by heat rest and immobilization
  - 3 Swelling usually without joint effusion
  - 4 Deformity and malalignment occurs as a result of irregular degeneration
- C Secondary Symptoms Radicular pains occur when joint changes

In the spine cause irritation of the spinal nerve roots

### Treatment

A General Measures Most of the general measures discussed for the treatment of rheumatoid arthritis are applicable here

1 Emphasis must be placed upon

2 Adequate diet with total calories adjusted to meet the patient's body needs. Weight reduction is very important in obese patients to help diminish stress on joints

3 Adequate rest and sleep. Avoidance of overfatigue is especially important

4 Avoidance of physical activity which would cause undue trauma to joints

5 Correct posture (see page 333)

### B Drug

1 Salicylates are indicated for the relief of pain in the case of rheumatoid arthritis (see page 315)

2 Thyroid extract may be indicated in the case of patients who have coexisting hypothyroidism

C For local treatment of joints (see page 333) Complete rest and immobilization of involved joints for short periods may be instituted without fear of complicating ankylosis although one must consider other harmful effects of bed rest in such patients (see page 2)

TABLE OF DIFFERENCES IN RESPONSE TO THERAPY

	Rheumatoid Arthritis	Osteoarthritis
Rest	Complete rest and immobilization attended by danger of ankylosis	Complete rest diminishes mobility of joints; is usually indicated for variable periods. Little danger of ankylosis
Exercise	Exercise should be encouraged in the convalescent phase of the disease	Mild exercise is desirable but undue exercise is harmful
Massage	Light massage over the joint may be indicated in the convalescent or chronic stage	Massage should be avoided directly over the bony overgrowths of the involved joint
Chiropractic	Often effective	No response. Not indicated
Physiotherapy	Often effective	No response. Not indicated
X-ray therapy	No response	Sometimes effective in relief of pain
Surgery	Often effective in relief of pain	Usually no response

## GONOCOCCAL ARTHRITIS (code No III 103)

A specific infectious arthritis caused by *Neisseria gonorrhoea* (gonococcus) occurring as a secondary complication of primary infection of the genitourinary tract or conjunctivae

### Diagnostic Features

History of previous genitourinary or ocular gonococcal infection and possibly of genitourinary trauma. Rheumatoid arthritis complicated by unrelated gonorrhoea occurs more commonly than gonococcal arthritis *per se*

#### A. Pariental Phase

1 Fever Mild to moderate Occasionally chills

2 Laboratory findings

a Leukocytosis Mild (10 000-15 000)

b Blood cultures Rarely positive

#### B. Arthritic Phase (Joint, Tendon and Bursa Involvement)

1 Early Evanescent polyarticular joint involvement of 3-7 days duration Joints red, warm, swollen and painful

2 Late Knees (74%), ankles (56%), feet (32%), wrists (15%) most frequently involved joints

a Joints initially red, warm, swollen and painful

b Ankylosis may occur in untreated cases

3 Laboratory findings

a Gonococcal complement fixation test: Doubtful value especially if positive since positive complement fixation tests are known to persist many years after genital infection

b Cultures of synovial fluid with special culture media are the most reliable method of diagnosis but are difficult to perform

### Treatment

A General Measures See general measures as discussed in management of rheumatoid arthritis (page 311) and physical measures in the management of the acute phase of involvement of the various joints (page 323)

B Specific Treatment Penicillin 25 000-50 000 units 4 times a day every 3 hours for 7-10 days. If improvement is not apparent in 3-4 days give intra-articular injections of penicillin 10 000-25 000 units daily into the larger involved joints

### BURSITIS

(Due to Infection) Acute (code No III 190)

Chronic (code No III 190 0)

(Due to Trauma) Acute (code No 25 4x0)

Chronic (code No 25 4x0 0)

(Due to Unknown Causes) (code No III 930)

Bursitis is an acute or chronic inflammation of any of the numerous bursae of the body. It may result from trauma, acute or chronic infection, or from unknown causes. Localized pain, tenderness and swelling may be observed at points around joints corresponding to anatomic bursae. Pain and limitation of motion of

adj = 1 joints are common Enlargement and calcification of bursae may be demonstrated radiologically 4 times

A General Measures Analgesics (see page 36)

B Local Measures

- 1 Rest and support of involved area by slings, splints, bandages, etc.
- 2 Local heat or cold Topical applications (see page 327)
- 3 Procaine hydrochloride solution 0.5-2.0% injection
- 4 Hydrocortisone acetate (Compound F) 10 mg has been reported to produce relief of acute bursitis when injected directly into the bursa
- 5 Aspiration of fluid from bursa Fluid should be examined
- 6 X-ray therapy in selected cases (by specialist)
- 7 Surgical removal in selected cases

### FIBROSITIS OR FIBROMYOSITIS

(Periarticular Fibrositis code No 34 x40)

(Chronic Myositis code No 33 190)

A loosely defined group of acute or chronic involvement of subcutaneous tissue, fibrous tissue of muscles and joint capsules, fasciculi, ligaments, tendons and fibrous connective tissues of certain peripheral structures due to a wide variety of causes most of which are not accurately determined. The condition may be manifested by pain, tenderness or stiffness of any involved portion of the body. Clinical and laboratory findings are minimal or absent.

A General Measures

- 1 Eliminate aggravating factors
- 2 Rest See section on physical management of joint disease page 323
- 3 Analgesics (see page 36)

B Local Measures

- 1 Local heat (see page 327)
- 2 Procaine hydrochloride 0.5-2.0% injection in trigger points (Of doubtful value)
- 3 X-ray therapy (by specialist) in selected cases which fail to respond to other therapy
- 4 Massage and graded exercise may be valuable
- 5 Stiffening of the involved structure responded by local heat or cold by procaine infiltration may give complete relief in some instances. If only partial relief is obtained, the procedure may be repeated daily.

### GOUT (code No 010 741)

A disease of unknown etiology characterized by recurrent attacks of acute inflammation due to deposition of sodium urate in the joints and in soft tissues throughout the body. Recurrent attacks of acute arthritis, separated by comparatively long intervals, is the most pathognomonic of gout, and the finding of uric acid crystals in the synovial fluid is diagnostic. An acute attack is usually self-limiting.

is very common even in asymptomatic periods. X-ray evidence of punched out areas about the joints is almost diagnostic but this occurs late

### Treatment of the Acute Attacks

#### A Specific Measures

- 1 Colchicine D S P B P is the drug of choice. It should be given as early as possible in the acute attack or during the prodromata to obtain maximum benefit. Give 0.5 mg ( $\frac{1}{2}$  120 gr) every 1 hour or 1 mg ( $\frac{1}{2}$  60 gr) every 2 hours until there is relief from pain or until nausea or diarrhea appear then stop the drug. The usual total dose to achieve this is 4-8 mg ( $\frac{1}{2}$  6-12 gr) and the pain and swelling will subside in 24-72 hours. Once the patient knows the dose that produces toxic symptoms the drug should be given in a single dose of about 1 mg ( $\frac{1}{2}$  60 gr) less than this. Then continue colchicine 0.5 mg ( $\frac{1}{2}$  120 gr) b i d q i d until attack has completely subsided. If diarrhea becomes too severe treat as for any acute diarrhea (see page 258).
- 2 Corticotropin (ACTH) and cortisone may provide dramatic symptomatic relief in acute episodes of gout but since colchicine seems to be about equally effective and provides a more lasting effect the latter still appears to be the drug of choice. It has been observed that when ACTH and cortisone are discontinued shortly after termination of attacks many patients will promptly relapse unless colchicine is given.

#### B General Measures

##### 1 Drugs

- a Analgesics. At times the pain of an acute attack may be so severe that relief of pain is necessary before colchicine becomes effective. In these cases codeine, with or without aspirin, may be given. Morphine should be avoided for fear of addiction in this chronic disease.
- b Cinchophen or neocinchophen should not be used.
- 2 Rest. Bed rest seems very important in the management of the acute attack. Bed rest should be continued for about 24 hours after the acute attack has completely subsided. Early ambulation may precipitate a recurrence.
- 3 Physical therapy is of little value during the acute attack although hot or cold compresses to the affected joints may make some patients more comfortable.

### Interim Treatment

A Specific Measures. Therapy aimed at prevention of acute attacks has been generally quite discouraging.

#### B General Measures

##### 1 Diet

- a Low purine. Most low purine diets (low weekly allowance of meat and avoidance of kidney, liver, sweetbreads, sardines, anchovies, meat extractives) tend to become nutritionally inadequate and often fail to influence the hyperuricemia or course of the disease. However, in gouty arthritis the restriction of high purine foods appears to be of great importance in prevention of progression of the disease.

- 1 Alcohol Alcohol has often been blamed as a precipitant of attacks. However there is little evidence that alcohol in moderation will do this or is at all harmful in this condition.
- 2 Colchicine The daily use of colchicine is controversial and the drug is rarely effective in preventing attacks. If it fails to reduce incidence of attacks it should be reserved for acute attacks.

### Treatment of Complications

- A Chronic Gouty Arthritis In recent years the outlook for patients with this disease has greatly improved. In many cases the progress of the disease is arrested and in some cases the absorption of gouty deposits may occur. This condition is best treated by a low purine diet and the new uricosuric drugs.
- 1 Uricosuric drugs
    - a. Probenecid (Benemid®) an agent which blocks the tubular reabsorption of filtrate in the kidney has been employed in doses of 0.5 Gm b.i.d. to q.i.d. for long periods and has been reported to provide relief in chronic gouty arthritis. Acute attacks of gout may occasionally be precipitated by this treatment but it is claimed that its administration is continued treatment. Full doses of colchicine may be used with Benemid®.
    - b. Sulfinpyrazone (Sulzylat®) Large doses of sulfinpyrazone (up to 3 Gm.) daily have been reported to produce a uricosuric effect similar to the above with relief of symptoms. Do not use concurrently with Benemid®.
    - c. Phenylbutazone (Butolidin®) A recent report indicates that it is effective in acute and chronic gout complicated by lowering of serum uric acid. The drug is administered orally as enteric coated tablets 100 mg. 3 tablets daily according to dose. Toxicity includes nausea, irritation of the stomach, vertigo, rash and dermatitis.
  - 2 Surgery may offer some help in relieving intractable forms but is not always effective.
- B Renal Complication The formation of uric acid in the kidney is a serious complication. The 1300 cc. of urine daily. Once all uric acid has formed little can be done to dissolve the stone although forcing fluids and alkalinizing the urine with 2-4 Gm (2-4 cc) of sodium lactate or sodium citrate per day may be helpful. The prevention of further stone formation.

## PHYSICAL MANAGEMENT OF ARTHRITIC JOINTS (PHYSICAL THERAPY)

### General Principles

Conservative principles which apply to the treatment of diseased joints are emphasized.

1 A range of support the firmest and most comfortable position which will provide for quiet rest. Use in the event that joint motion be subsequently lost.

2 In the early stages of arthritis after the acute process

has subsided employ careful active exercises or passive mobilization early and regularly as tolerated in order to prevent deformity and to preserve joint motion

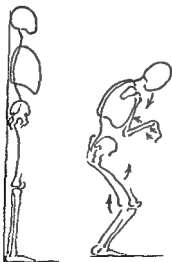
3 Avoid measures which cause a persistent increase in symptoms. So called routine measures e.g. heat are not uniformly tolerated by patients. The correct balance of heat, massage, rest and exercise must be planned for each patient.

4 Patients with joint disease (particularly rheumatoid or suppurative arthritis) are in constant threat of deformity. Guard particularly against flexion deformities

5 The services of a specialist in physical therapy should be utilized whenever possible

6 If the arthritis is severe the course of the disease seems unfavorable or ankylosis appears inevitable early consultation with a specialist is imperative. Special orthopedic measures such as manipulation under anesthesia, traction, special casts, braces and corsets and surgical measures including arthroplasty, capsulectomy, tenotomy, arthrodesis and synovectomy may be required.

7 Emphasize to the patient the importance of complete co-operation and his responsibility with the physical therapy program at home as well as in the office or hospital. Stress the importance of year round continuance of treatment if necessary. Instruct the patient and/or his family and friends as to the exact and proper use of heat, immobilization and passive mobilization under home conditions.



Normal Posture and the Deforming Tendencies of Joint Disease

## OPTIMAL FUNCTIONAL POSITION OF JOINTS

Should ankylosis set in inevitable despite of quasi therapy of desirable early orthopedic consultation is imperative. The following table gives certain commonly accepted optimal functional position in which joints may be permitted to fuse. It must be emphasized however that a given position is functional for a given individual depending upon such factors as living habits, occupation, reaction and personal preferences.

Joint	Position
Shoulder	Arm abducted at about 75°. The elbow joint placed in line with the anterior chest and with partial pronation of the forearm.
Elbow	Elbow at slightly less than 90° with the forearm in a position midway between supination and pronation. Ex-pilone laborers fill supination; clerical workers fill pronation.
Wrist	Slight (30°) extension.
Hand	Partial flexion of fingers and metacarpophalangeal joints and partial position of thumb.
Hip	Unilateral in extension with minimal abduction and minimal lateral rotation. Bilateral involvement: Slight flexion of one hip joint and the other abducted.
Knee	At the individual's full extension. Sedentary individuals 35 of flexion. Young individuals with active epiphyses: Only light flexion.
Ankle	Foot at 90° angle with leg minimally extended.

## REST IMMOBILIZATION AND SUPPORT

### Spine

Rest in a comfortable position on a flat firm bed without pillow at head is essential. A 3-5 ft plywood or other impervious type of board placed under a thin, firm hair or felt mattress and immobilization and support of the spine by simple well applied adhesive taping may give prompt temporary relief. Suitable rest or brace may be employed on ambulatory patients with mild symptoms. Special body molds (plaster shell) or rigid jackets may be advised by the orthopedist for patient confined to bed.

### Cervical Spine

Rest in comfortable position on a flat firm bed without a pillow (above). Immobilization and support of the head may be accomplished by special orthopedic or home-made collar. The latter may be made simply by folding soft cloth two or three times lengthwise and wrapping snugly around neck and fastening with pins. Traction may be necessary if deformity, pressure or pain to nerve



## 326 Immobilization

### Shoulder

Rest in bed in a comfortable position on a flat firm bed without a pillow (as above) Support the arms with pillows in a position of intermediate abduction and external rotation After patient is ambulatory the arm of the involved shoulder may be supported by a sling

### Elbow

Support the arm and hand (thumb and fingers free) in a molded bivalve plaster cast with the elbow in a position of maximum tolerated extension This is to combat the natural flexion tendency

### Wrist

Provide rest and support for the hand in a bivalve splint which corrects the natural deforming tendency to wrist flexion and ulnar deviation At first the splint should be worn continuously except for removal 1 or 2 times daily to permit physical therapy later the splint need be worn only in bed

### Hand

A supporting plaster splint fitted into the palm and extending to form a pocket for the partially flexed fingers may prevent the natural deforming tendency of hyperextension of the metacarpophalangeal and distal interphalangeal joints and flexion of the proximal interphalangeal joints This splint should be removed 2 to 3 times daily to permit physical therapy

### Hip

The patient should be at bed rest A detachable plaster hip spica may be used to provide support and rest for the acutely involved hip joint It may be worn all night but it must be removed at least 2 or 3 times daily to permit physical therapy (at least in the case of rheumatoid joints) The patient is instructed to lie prone in bed with 1 or 2 pillows (as necessary) under the abdomen to fix pelvis for  $\frac{1}{2}$  to 1 hour 1 or 2 times daily (The weight of the body is utilized as a load against the powerful flexor muscles of the thighs) The pillows may be removed as tolerated and as the flexion deformity is corrected

### Knee

The patient should initially be at bed rest Weight bearing upon the acute joint should be restricted or prohibited In mild and non deformed joints the posterior plaster splint is convenient to use and will suffice It should be worn almost continuously while the patient is in bed particularly during the night Adjustable splints with slight flexion may be employed on patients who are able to walk When joint involvement is more marked and flexion deformity is present correction plaster casts are applied in a position of maximum correction and left on for 3 days The cast is bivalved and removed for physical therapy twice a day New casts are made to provide further correction as indicated During convalescent or chronic phase provide support for the knee with elastic bandage posterior splint or special orthopedic braces

### Ankles and Feet

Weight bearing upon the acutely involved joints must be

prohibited. Provide a cradle or large pillow at the foot of the bed under to hold the bed both a off the feet. A supporting removable plaster boot (with tip of toes exposed) is available. Adjustable or arial bivalved plaster foot casts may be employed for the gradual restoration of deformities. Provide well built shoes allowing proper length and width for toes, stability and a suitable arch support (sponge rubber or felt pads are quite suitable to y). Correct abnormalities and deformities of the hip and knee joints which produce mechanical faults in the feet.

## HEAT

### Local Heat

#### A General Principles

1. Place the part of the body to be treated in a comfortable and relaxed position.
2. Begin treatment slowly and cautiously.
  - a. Treat for short periods not longer than 15 to 20 minutes initially. When the skin is pink and moist enough heat has been given.
  - b. Start with low temperature and adjust it according to individual tolerance.
3. Gradually increase time and temperature factors as tolerated and as indicated.
4. Avoid drafts in the treatment room.
5. Following treatments provide protection over it for 20 to 30 minutes to avoid chilling.

#### Indications

1. Acute and chronic diseases of joints, muscles, fasciae, tendons and bursa to relieve pain and to reduce muscle spasm.
2. Abdominal cramp (non urgent abdominal).
3. Chronic involvement of joint muscles, fasciae, tendons and bursa, to relieve pain, reduce muscle spasm, hasten recovery process and to serve as an adjunct or preparation for other physical therapy methods.

#### Contraindications

1. The temperature in whom local heat is contraindicated and possibly aggravates symptoms (if the patient has a trial of cold therapy may be warranted).
2. Local diseases of the skin.
3. Peripheral vascular diseases (vascular insufficiency) (see page 307).
4. Diabetic patients (use cautiously).
5. Patients with loss of skin sensation.
6. When large areas are to be treated.

#### Methods

1. Conductive heat. Heat transferred to the body by direct continuity with the source. This is the most penetrating form of heat.
  - a. Hot water bottle or bag or local heating pad. Simple and readily available heat source. There is some danger of cutaneous burns.
  - b. Hot compresses. Simple readily available and effective.

both home and hospital use. The water should be maintained between 98° to 103° F (36.5 and 40.3° C) the bath should be for 10 to 15 minutes as tolerated.

- 2 **Wet packs** An effective technic for hospitals but not generally satisfactory for home use. Cold wet sheets are carefully applied to the skin and the patient is wrapped in blankets. The patient is allowed to remain in this pack for 1/2 to 1 hour. This method should never be employed by untrained personnel.
- 3 **Steam baths** Not advisable for home use since home made steam baths may be dangerous.
- 4 **Body baker** A larger version of the ordinary baker described on page 329 may be employed on patients who are in the recumbent position. For patients who are able to sit up cabinets with numerous radiant electric lamps or resistance coils may be used.
- 5 **Sun baths (heliotherapy)** Graded daily exposure (as tolerated) to the sun's rays is beneficial for its combined heating, ultraviolet and tonic effects.

## COLD

In rare cases patients are unable to tolerate local heat and will do well with local applications of cold.

- A **Cold compresses or ice packs** applied locally to the joint for 10 to 15 minutes 3 or 4 times daily as needed.
- B **Ethyl Chloride Spray** Particularly indicated in fibrositis or osteoarthritis. It is applied like local anesthesia to the trigger point as a substitute for procaine infiltration or heat. Ethyl chloride spray should be followed by stretching and exercise.

## MOBILIZATION OF JOINTS AND SURROUNDING SOFT TISSUES

After the acute process has subsided institute exercise as early as tolerated in order to prevent deformity, muscle atrophy and altered joint motion (see page 324). No other physical therapy methods will prevent these abnormalities or serve as a substitute for exercise.

Proceed cautiously with graded exercises. Avoid sudden transitions. Reduce the intensity or change the nature of the exercise if there is persistence of or increase in symptoms (spasm and pain).

### Passive Exercise

- A **Simple Passive Motion of Joint Through the Existing Range**  
This is done by a physical therapist or other person or by the patient himself. The movement is slow. It should cover the complete available range of the joint and be repeated several times. No force should be used and the patient should be relaxed. The objective of passive motion is to prevent loss of range of motion, particularly in patients who are immobilized by splints, slings and bags, etc.
- B **Stretching** Similar to passive motion and is somewhat forceful.

it is c i d slightly beyond the e lating ange This p oced re m y caus a ce tain am unt of pain and should be p e ded by analg sic (a e p g 38) Stret hing sho be pe formed only by a physical th rapt or physician The i dications fr t tch ing are as follows

- 1 In d f rming types of arthritis It is us d t d c ea e fl xion contr ctur
- 2 In o t ythritis and f brostit It m y act lly le d t a cu by giving omplet pain ll f
- 3 In p ture c r ction stret hing t an adjunct to active exercia

C Manip lation A p sive rath r f ful a d s dden mobili s [on / ] int in dir tion whi h is ot used physi logi ally e g sid w d motion or ot tion in a m t erpoph ang l ; int Manipul tion should be don only by a phy i ill pr f bly without anesthesia Manip lation is used t bre ll us pai f i l t rcul adhesion It may al be u ed in te d f t t h g lth th d vant g that th mobili zation cannot be ouniera ted by voluntary out tions o spasm be au of the di tion of th p l

## M s g

L ntially mobili tion of oft tis ve by dir t m l or digit l action M ssg i f rmo huc in ll m toid arth itis It is f more val in fibrostit but an e dily be r pla d by t et h g and e l Cont ndi ations fo mas ge are local skin di phl bitt and ad an ed r l osclerosis Se l typ have b desc bed

- A Stroking ( fflen age) ha ntially p y hologi l and anal ge l tff l
- B Kn dng (petr e g ) prod di t m ualst t h of mu l and a butan oast ti ve It i and t d in fib ostit wh th masse m y f l e d rub out p inf l f brostitic ond l s
- C F i tion m g ron lating f small c lar movem ns of th fing r tips will e ve th me p rpos
- D P us t nd fbr ting mo m t are ot f medi l al

## Et t of The r

- A Number of Sess The g St t hing and m l nce of r nge of motion may be p es ibed as fr que tly a very 2 huc M s g usually not given more than on e a day manip lation not m than on o twice a we k The minim m of the py de pend on the pati s cond tion A tagl t t t ing or mani p lation m y cu a fibrostit In deforming joint d eas th mobili zation should be f que t nought f ll ob tive of m lnt ining or increas g ge of motion
- B Duration of T m M sive motion and t tching should be pr d sere t times for e ch j unt l or y out j unt is involved the proc du sh ll last only 2 to 3 mnd s l sere al joint ret t d st will at large but how d not eed l s mnd s If all joints shoud be t at d in one s s ion the t rme of tr time to hould be huc d and the joint t r rated

rapidly as possible

- G Still Bars (ladder exercises) For girdles and extremity joints
- H Treadle Exercises for ankles and knees
- I Pedaling Exercises (bicycle exercises) For hips and knees
- J Stepping for lower extremity joints
- K Special quadriceps exercises, which include static contraction straight leg raising active motion and graded resistive exercises

#### Occupational Therapy or Recreational Exercises

Provide an incentive for the patient to use impaired parts. These measures may be instituted by trained therapists in the hospital or in the home and the instructed patient may carry out the therapeutic program. The desired objectives, the precautions and the limitations of such methods must be explained carefully to the patient. Lack of special facilities or equipment can be more than readily compensated for by some thought and ingenuity in utilizing materials at hand. For example, the forearms, wrists and hands may be exercised effectively by typing, piano playing, string instrument playing, business machine operation, molding or clay modeling, wood and machine shop work, weaving, wood carving, needlework and painting.

## Chapter 13

# DISEASES OF THE NERVOUS SYSTEM

## DISORDERS OF CONSCIOUSNESS

Disturbances of the cerebrum may be associated with decreased motor activity (e.g. stupor or coma) or increased motor activity (e.g. excitement of delirium mania). Sopor is a disturbance which may result from partial clouding of consciousness to complete obliteration of consciousness. The point of reaction of these disorders depends upon the nature of the stimulus and the physical mental and emotional status of the individual. Some of the causes of coma are: severe cerebral injury, cerebral degeneration, poisoning (e.g. morphine, barbiturates, digitalis), overwhelming infection, convulsive disorders and renal decomposition.

### STUPOR (code No. 933) and COMA (code No. 932)

Stupor is a condition of partial to almost complete loss of consciousness. Coma is complete loss of consciousness from which the patient cannot be aroused by the most powerful stimuli.

#### Differential

- A. History - Is the patient during the intervals Valuable information may be obtained from the patient's relatives and attendants. Inquire particularly about the patient's occupation, previous physical, mental or emotional illness and about use of alcohol, drugs, epilepsy or hypertension.
- B. Physical Examination - Place particular emphasis on vital signs and evidence of injury or intoxication and neurological abnormalities. Do not assume cerebral disturbance due to alcohol or any other cause until a definite cause has been detected.
- C. Laboratory Procedures -
  1. Cerebrospinal fluid - the patient if necessary and examine the serum for electrolytes, albumin, blood sugar and ketone.
  2. Urine - examine for Hgb, WBC, d.t. and other abnormalities.
  3. Blood - for N.P.N., glucose and  $CO_2$  combining power which indicate for diagnosis of diabetic coma and uremia.
  4. Lumbar puncture should be considered for all comatose patients unless there are specific contraindications (e.g. severe degenerative diseases).
  5. Special studies may be indicated, e.g. blood gases and

- analysis of body fluids for evidence of toxins
- 6 Skull x rays when indicated

### Treatment

**A Emergency Measures** The objective is to maintain life until specific diagnosis is made and treatment administered

- 1 Maintain adequate respiration First determine the cause of any respiratory difficulty (e.g. obstruction pulmonary disease depression of respiratory center vascular collapse)
  - a Keep airways open Obstruction must be removed or prevented
    - (1) Place patient on his side or abdomen with face to the side always with the head well extended lower on his back or with head flexed If necessary pull tongue forward with fingers or forceps and maintain in an extended position (e.g. by pharyngeal airways)
    - (2) Aspirate mucus blood and saliva from the mouth and nose by means of a lubricated soft rubber catheter Suction may be conveniently applied with a large (25-50 cc) syringe
    - (3) Endotracheal catheterization may be necessary The services of a trained anesthetologist or otolaryngologist are desirable for this
  - b Artificial respiration may be administered if respirations have ceased or are failing (see page 133)
  - c Oxygen may be administered by mask catheter or tent as indicated (see page 139)
- 2 CIRC Institute immediate treatment if patient is in shock or may suffer shock (see page 31)

### **B General Measures**

- 1 Constant observation of the patient must be maintained
- 2 Place in shock position unless this is contraindicated by head injury (see page 3) Change body positions every 1/2-1 hour unless contraindicated to prevent hypostatic pneumonia and skin ulcerations
- 3 Catheterize patient if coma persists for longer than 8 to 12 hours and patient fails to void If necessary insert an indwelling catheter Use sterile technic
- 4 Nutrition and hydration Provide proper fluid and nutrition by I.V. glucose amino acids and saline solutions (see page 27) for the first few days until the patient is able to take fluids by mouth If the patient is comatose for more than 2-3 days tube feedings must be employed (see page 57)
- 5 Sedation
  - a When ever possible avoid sedation or other medication until a specific diagnosis has been made
  - b Sedation with paraldehyde or barbiturates may be necessary for mild restlessness in those cases not due to barbiturate or other drug toxicity

**C Specific Measures** Direct measures at removal of specific causes such as fevers infections toxins (see specific diseases)

# DELIRIUM (code No 931) and MANIA (code No 037)

Delirium is characterized by marked disturbances of consciousness and hallucinations (visual, auditory, tactile, olfactory, gustatory, and somatic) physical symptoms associated with the delirious state of consciousness.

Mania is a form of insanity often temporary characterized by wild or raptidity and at times by illusion, delusions and hallucinations.

These two conditions are discussed together because they share many points in common. The principal therapeutic difference lies in the choice of sedative and hypnotic medications. Although most sedative and hypnotic drugs in proper dosage may be used with relative impunity in mania, the number of drugs which can be employed in delirium is limited. It is advisable to restict the drugs for delirium to paraldehyde, chloral hydrate and in certain cases opolamine or a coti. Chloral hydrate is contraindicated in acute alcoholic delirium.

## Discussion

See Com. page 335

## Treatment

### A. Patient From Physical Illness

1. Quarters: Clean, comfortable room available preferably on lower floor of building.
2. Windows: Screened or otherwise protect windows. Lock down the curtains to avoid glare.
3. Furniture: Remove all furniture and furnishings from the room except a low bed with side boards or at least simply mattress on floor. The room must be free of sharp objects.
4. Avoid mechanical stimuli which may precipitate epileptic or epileptiform mediators or surgical and chemical stimuli such as paraldehyde or chloral hydrate or use hydrotherapy as mentioned later. Observe for suicidal or destructive tendencies.

### B. Psychological Patient

1. Be kind and understanding. Recognize patient's reaction to those of confusion and hyperemotion. Do not withdraw.
2. Lighting and noise: See that the room is adequately lighted both day and night and free from shadows. Loud noises should be avoided but familiar sounds are actually soothing to the patient. Remember that the patient may be over-sensitive to stimuli.
3. Help the patient to understand what is happening and why he is in hospital. Inform him of the patient's present position. Explain the grounds and the appropriate procedure when necessary.
4. Relatives and friends: Recognize the patient's relationship to the patient's family. They may have a right to know the patient's condition. How to handle the patient's family is often a frequent problem. The patient's family should be informed of the situation.
5. Consider the patient's individual needs.

### C. Use of Hypnotic Drugs

1. For the purpose of inducing sleep, the drug of



choice in delirium. Barbiturates, bromides, and opiates often serve to increase the excitement of delirium but may be used in maniacal states (see below). Paraldehyde has an added advantage in that the ordinary stock paraldehyde solution needs no sterilization and for that reason is available for immediate administration by any desired route. The oral route is preferred unless the patient is unable to swallow. For details of administration see page III.

- 2 Chloral hydrate may be given instead of paraldehyde in doses of 2 to 8 cc ( $\frac{1}{2}$  to 2 dr.) of the 25% stock solution or as capsules 0.3 to 0.6 Gm ( $\frac{1}{2}$  to 30 gr.) orally. Chloral hydrate is contraindicated in acute alcoholic delirium or psychosis.
- 3 Barbiturates. Not to be used for delirium.  
Caution. Observe carefully for respiratory depression and see that adequate airway is maintained.
  - a Thiopental Sodium U.S.P. Thiopentone Sodium B.P. (Pentothal Sodium®). First inject 2 to 3 cc of a freshly prepared 3% solution slowly I.V. observe then give additional dosage as needed for desired effect.
  - b Amobarbital Sodium (Amytal Sodium®) N.F. 0.125 to 0.5 Gm (2 to  $\frac{1}{2}$  gr.) as freshly prepared 10% solution slowly I.V. to point of desired effect.
- 4 Morphine sulfate 8 to 15 mg ( $\frac{1}{8}$  to  $\frac{1}{4}$  gr.) with scopolamine hydrobromide 0.3 to 0.6 mg ( $\frac{1}{2}$  to 1  $\frac{1}{2}$  gr.) may be administered subcut. when delirium is marked or is associated with or caused by pain.
- 5 Scopolamine hydrobromide. For delirium without pain. Scopolamine 0.3 to 0.6 mg ( $\frac{1}{2}$  to 1  $\frac{1}{2}$  gr.) b.i.d. q.i.d. may be valuable.

#### D Hydrotherapy

- 1 A warm tub bath (92° to 97° F.) or so called neutral bath for half hour periods t.i.d. or q.i.d. may be tried on suitable patients. This may be of considerable value. This method should be tried prior to intensive drug therapy whenever possible. If it is tolerated well and results are effective the patient may remain in the tub for hours. Hydrotherapy is not applicable for certain unmanageable patients, for patients with infectious or febrile diseases, or for patients with surgical dressings.
- 2 Wet pack. This effective technic should be administered only by trained personnel. The patient requires constant supervision. The method is contraindicated in patients who are physically weak or exhausted or are having convulsions or who have significant cardiovascular disease. Vital signs must be observed at least at 20 minute intervals.

E Nutrition and Hydration. Unless there is a specific indication for hypohydration, a normal state of hydration should be maintained. This is especially true in the presence of fever. For delirium tremens or alcoholism, 1 to 2 liters (1 to 2 qt.) of 5 to 10% glucose solution containing 100 mg ( $\frac{1}{2}$  gr.) of Thiamine Hydrochloride U.S.P. Anserine Hydrochloride B.P. and 100 mg nicotinic acid should be given daily. Proper nutrition should be maintained. Small frequent feedings are best tolerated.

**Psychiatric** If no danger sure attention above  
do not if consider transfer to psychiatric hospital. E  
statement of ability of effecting the transfer decided upon  
provide for adequate attendance

## HEAD INJURIES

Proper management of the patient with a head injury rests in  
great measure upon neurologic and surgical diagnostic and treatment methods

### Diagnosis

1. Initial examination and close observation of the patient in  
the immediate post-traumatic period are essential

#### A Signs and Symptoms

1. Alteration in the state of consciousness in the immediate  
period after the head injury  
A. A sudden fall followed by loss of consciousness may indicate cerebral  
impairment by a blood vessel or epidural hemorrhage. If pro-  
gressively deepening, may occur after a period of conscious-  
ness following a head injury, especially with a fracture of the skull.  
Indicates a serious condition, but not necessarily fatal.
2. Pupil size and reaction may indicate a subdural hemorrhage  
a. Ipsilateral pupil is usually dilated  
b. Contralateral hemiparesis may occur ipsilaterally to the  
pupil, but may also occur bilaterally
3. If the patient remains unconscious, diagnosis of prog-  
ressive intracranial hemorrhagic lesion is difficult  
a. Vital signs (pulse, rate, respiration, blood pressure) may  
fluctuate although the respiration is usually normal  
b. If a fracture of the skull is present, a depression of the  
skull may indicate a fracture of the skull  
c. If a fracture of the skull is present, it indicates a fracture  
of the skull and the brain

#### B Laboratory Findings

1. Lumbar puncture is inadvisable to establish the presence of  
subarachnoid hemorrhage and to give a line of reference  
and pressure of the cerebrospinal fluid
2. Skull x-rays should be made in those cases in which a  
fracture of the skull is suspected  
a. If a fracture of the skull is present, it may be detected  
b. Presence of a fracture of the skull may be certain
3. Examination of the patient may assist in diagnosis and prognosis  
of a patient with a head injury

### Treatment

#### A First Aid

1. If a patient is unconscious, a patient may be required (see page 12)  
2. Attention to the respiratory system is important. Main-  
tain a clear airway by turning the head to the side  
If a patient is unconscious, a patient may be required  
turned to one side to facilitate the flow of air from  
mouth and to keep the tongue from obstructing the pharynx

## 340 Orthostatic Hypotension

- Intratracheal intubation or tracheotomy may be necessary to maintain open airway
- Give oxygen if necessary (see page 145)

### B C n r a l M a s s a g e

- 1 Quieting patient During acute or initial phases restlessness may be a disturbing factor
  - Special nursing care and paraldehyde may be required
  - Avoid morphine because of medullary depressant effects
  - Catheterization of a full bladder may ameliorate restlessness
  - d Lumbar puncture with removal of small amount of bloody cerebrospinal fluid may also relieve agitated patient
- 2 Antibiotic treatment is always instituted in the presence of bleeding or discharge from nose or ears Give penicillin procaine 300 000 units b i d until danger of infection is over

## SYNCOPE (Fainting) (code No 0xx)

Syncope is a transient loss of consciousness due usually to temporary cerebral anoxia. The exact mechanism of syncope is not clearly understood.

### REFLEX SYNCOPE

#### VASODEPRESSOR SYNCOPE

(Vasovagal Syncope Simple Fainting Benign Faint)

This is usually characterized by a sudden fall in blood pressure and a slowing of the heart. The causative stimuli may be sensory (e.g. sudden pain) or entirely emotional (e.g. death of a loved one). The patient is usually upright when the faint occurs. recumbency rapidly restores consciousness.

#### Treatment

Patient should be placed in the recumbent position and head lowered. Simple inhalation of fumes of Aromatic Spirits of Ammonia U.S.P. B.P. may be tried if necessary.

### ORTHOSTATIC HYPOTENSION

(Postural Hypotension) (code No 450 x10)

This is a rare cause of syncope and occurs as the patient assumes an upright position. It is associated with a marked drop in blood pressure on arising.

#### Treatment

Treatment is directed towards the underlying cause where possible. If abdominal ptosis is present an abdominal belt may prevent splanchnic pooling of blood. Elastic stockings may be of value.

Vasoconstrictor drugs may be tried but are usually without benefit

### CAROTID SINUS SYNCOPE (code No 408 584.x)

There is usually a history of fainting associated with spells of dizziness between attacks. A definite relation to sudden turning or raising of head or wearing of a tight collar may be elicited. The diagnosis is suggested by reproducing it by firm pressure and massage over the carotid sinus for 10 to 20 seconds. Stimulate only one carotid sinus at a time. Caution must be exercised in stimulating the sinuses in elderly patients. Cerebrovascular accidents have been precipitated by this maneuver. Three types of carotid sinus syncope are known to occur

#### Vagal Type

This is the most common type and is most frequent in old persons. Carotid sinus pressure slows the heart rate. This response can be abolished by the injection of Atropine 1 mg. U.S.P. B.P. 1 mg. (1/80 gr.) i.v.

#### Vasomotor Type

Occurs more frequently in younger individuals. Carotid sinus pressure causes a fall in blood pressure; this can be abolished by injection of 0.5 cc (8 gr.) of 1:1000 Epinephrine in U.S.P. Adrenaline. B.P. but is unaffected by atropine sulfate.

#### Cerebral Type

Carotid sinus pressure affects neither heart rate nor blood pressure and neither epinephrine or atropine is effective in the relief. A direct cerebral effect is postulated.

#### Treatment

Correct all abnormalities whenever possible. Eliminate emotional problems and forbid use of tight collars. In severe cases denervation of the sinus may be necessary. Local anesthetic of the carotid sinus abolishes all type of carotid sinus syncope.

A Vagal Type Atropine sulfate 0.4 to 0.6 mg. (1/60 to 1/100 gr.) 3 to 4 times daily (more if needed) will usually abolish attacks. Ephedrine sulfate or hydrochloride 0.25 Gm. (3/8 gr.) with phenobarbital 0.015 Gm. (1/4 gr.) 3 to 4 times daily may be helpful.

B Vasomotor Type Ephedrine and phenobarbital as above will usually prevent attacks.

C Cerebral Type Drug are of no value.

### SYNCOPE DUE TO CARDIOVASCULAR DISORDERS

This type of syncope is due to cerebral anoxia which has its commonest temporary fall in cardiac output. Some of the causes are Stokes-Adams syndrome, onset of myocardial infarction, coronary artery infarction and pulmonary embolism. This may be associated with heart in other types of heart disease (e.g. aortic stenosis and tetralogy of Fallot).

Treatment.

Treat the underlying abnormality

**SYNCOPE DUE TO METABOLIC DISTURBANCES**

Hypoglycemia may cause syncope or coma. If prolonged or recurrent treatment is required (see page 409)

Hyperventilation if severe and prolonged produces respiratory alkalosis with resulting tetany and syncope

Treatment.

Consciousness can be restored by rebreathing into a paper bag holding breath or administration of carbon dioxide 5-10% with oxygen by mask. If attacks are recurrent psychotherapy must be considered

**HYSTERICAL SYNCOPE**

Hysterical fainting may either be true or simulated syncope. The physician must be watchful of the associated objective findings. The patient rarely if ever has an attack without the benefit of an audience

Treatment.

Psychiatric evaluation and psychotherapy

**VERTIGO AND DIZZINESS**

The term vertigo is generally used to denote the subjective sensation of rotatory movement either of the individual or his environment. Dizziness implies an inability to orient the body in relation to surrounding objects. However the terms are generally employed as synonyms

**TRUE VERTIGO**

This is found primarily in disease processes involving the labyrinth, the vestibular portion of the 8th cranial nerve and their nuclei or connections. True vertigo is usually manifested by nystagmus falling to one side and abnormal reaction to tests of vestibular function. Among the more common causes are

1. Meniere's syndrome (see page 357)
2. Acute labyrinthitis (see page 357)
3. Organic brain damage involving the vestibular nerve, its end organs or connections or the cerebellum
4. Drugs and toxins (e.g. streptomycin see page 507)

Treatment.

Treat the underlying disorder

## DIZZINESS

Dizziness is a subjective complaint. There may be no objective findings; it may, however, be associated with infectious processes and other toxic conditions. It also occurs in cases of cerebral vascular disease with impaired circulation and is a common symptom in hypertension. However, it is probably most often found in patients with emotional disorders.

### Treatment.

Restoration of optimum general health. If functional elements are present and the symptom is severe, psychotherapy may be of value.

## HEADACHE (code No 961)

Headache may be due to many factors and must always be recognized as a symptom. The underlying cause must be determined and treated in order to effectually relieve the symptom. The subjective sensation of headache indicates involvement of the pain-sensitive structures within and about the skull. Headaches may be classified as follows, with some of the more common causes listed:

- A Meningitis (and Allied Stricture Involvement). This is due to suppurative infections involving the meninges and intracranial pressure and is characterized by intracranial pressure (following lumbar puncture).
- B Vasomotor Headache.
  - 1 Intercranial vasodilatation. Due to fever, reaction to drugs and toxins (e.g., alcohol and histamine), or is of emotional origin.
  - 2 Extracranial vasodilatation (particularly of the external carotid). Migraine is the principal example.
  - 3 Disease of the blood vessels (e.g., temporal arteritis).
- C Musculoskeletal Involvement.
  - 1 Muscular pain of varying degree. Due to myositis, adjacent arthritis, or osteitis.
  - 2 Muscle tension due to emotional factor.
  - 3 Bone or joint involvement of skull, head, or cervical vertebrae. Due to arthritis, osteitis, osteomyelitis, or tumor.
- D Neuralgia. Headache (e.g., trigeminal neuralgia).
- E Middle Ear Infection Involvement. Due to disease and discharge of the middle ear, nasopharynx, teeth, etc.
- F Emotional Involvement. Headache due to motion disorders are usually associated with muscle tension (see above), but this is not always the case. At times the headache may be due to intracranial vasodilatation.

### Diagnosis

The diagnosis must be based on a complete history and physical examination. Special attention to the eyes, ears, and nose is important. A complete blood count, urinalysis, and blood test for syphilis must be performed. In many cases an adequate psychiatric examination is indicated. Skull x-rays and other special studies are as follows:

## 344 Headaches

The pain of meningeal involvement is deep and is usually the most severe. Pain of vascular origin is usually throbbing in character. Pain of neuralgia has a burning quality. Headaches of psychogenic origin are superficial and are manifested by dull tightness or pressure.

### General Nonspecific Treatment Measures

- A Physical and mental rest
- B Sedatives should be used only as a temporary measure and should not be used as a substitute for a complete work up and specific therapy. Narcotics are generally contraindicated except in terminal disease.
- C Analgesics constitute specific therapy in febrile headaches due to their antipyretic activity. They should not be administered for prolonged periods indiscriminately; their routine use often obscures important pathology.

## HEADACHES DUE TO MENINGEAL INVOLVEMENT

These are the most severe, but they usually respond to analgesics. Manifestations depend upon type and site of underlying pathology.

### Treatment

A Specific Measures. Treat the causative lesion.

B General Measures

- 1 Analgesics should be given as needed if pain is not too severe (see page 36).
- 2 Narcotics may be necessary if pain is very severe (see page 37).
- 3 Lumbar puncture performed very cautiously may sometimes be used to relieve headache associated with increased intracranial pressure (e.g., subarachnoid hemorrhage, hypertension, nephritis, not in posterior fossa tumors).

C Lumbar Puncture Headaches. These are believed to be due to leakage of the cerebrospinal fluid from the puncture site.

- 1 Analgesics. If headache is mild upon arising, analgesics such as aspirin 0.3 Gm (3 gr) every 2-3 hours may be sufficient.
- 2 Recumbent position. If lumbar puncture headache is very severe, it can be alleviated by lying down.
- 3 Intrathecal injection of small quantities of sterile normal saline may afford relief in severe cases.

## HEADACHES DUE TO VASCULAR INVOLVEMENT

These headaches are usually throbbing in character. Intracranial vasodilatation usually causes bilateral pain, but migraine, an extracranial type, is usually unilateral. Compression of the common carotid may relieve both types of headaches; migraine may also be relieved by compression of the external carotid artery.

Tr atm t  
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drug as e ly in attack as possibl Do not r peat  
dose more oft n than once w ekly

(2) E got mine tart at by mouth 4 to 5 mg (1/5 1/2 g )  
s blingually o orally continue with 2 mg (1/20 g )  
very hour until he dach h dis appeared or until  
total of 11 mg (1/5 gr ) has be n administ ed This  
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Drugs  
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(2) E gotamin ts t 3 4 mg (1/20 1/15 g ) sublingu lly  
(3) Aspi l 0 6 Gm (10 gr ) w th or w thout odeline  
0 06 Gm (1 g ) by mouth may be us l in mild  
att k

re ve tion of further att k M gr ne s a p ychosom ti  
a R duction in at k m y be a onp li hed by



psychotherapy There is little evidence that special diets glandular therapy etc are effective except as psychotherapeutic devices

- C Disease of Blood Vessels Since many of these conditions are associated with vasoospastic phenomena vasodilators are indicated Nicotinic acid 100 mg (1½ gr) t i d to q i d orally has been found to be of limited value

## HEADACHES DUE TO MUSCULOSKELETAL INVOLVEMENT

Muscle contraction or spasm may be caused by disease of the muscle or adjacent structures or may be associated with excessive fatigue or emotional tension The muscles attached to the occiput are most frequently involved and give the characteristic occipital headache There may also be a feeling of pressure or tightness or a band like constriction of the head associated with emotional tension The psychogenic headache usually appears after periods of emotional stress

### Treatment

- A Muscle spasm due to organic disease and bone or joint pain may be relieved by appropriate physical therapeutic measures (see page 323) Analgesics are usually also of value (see page 36) Specific therapy should be directed at the underlying disease
- B Muscle Tension Headaches
- 1 Rest relaxation and freedom from emotional stress are of primary importance (see Psychotherapy on page 41)
  - 2 Heat to the involved muscles by means of hot towels heat ing pad or a warm bath will help relieve the discomfort
  - 3 Gentle massage of the muscles will usually also be of benefit
  - 4 Drugs may be of value in acute cases but prolonged use should be avoided
    - a Phenobarbital phenobarbitone 15-30 mg (¼-½ gr) q i d will temporarily relieve many headaches due to nervous tension
    - b Aspirin 0.3-0.6 Gm (5-10 gr) every 3 to 4 hours may also be of benefit

## HEADACHES DUE TO NERVE INVOLVEMENT

Treat specific cause (see page 356)

## HEADACHES DUE TO MISCELLANEOUS EXTRACRANIAL CAUSES

Treat specific underlying disease Analgesics may be of value but should not be substituted for specific therapy

## THE DEGENERATIVE DISEASES

## MULTIPLE SCLEROSIS (code No 906 953)

A disease of unknown etiology characterized by patchy demyelination in the central nervous system which may be due to or associated with diffuse vascular thrombosis. It is manifested by diffuse neurologic disturbances: trouble with neuritis, nystagmus, slurred speech, intention tremor and optic atrophy. S F examination shows nothing characteristic. The disease is slowly progressive with spontaneous temporary remissions.

Treatment

Chiefly symptomatic

- A Rest. Adequate sleep at night and rest in the afternoon has been found to make patients more comfortable.
- B Temperature Changes. A sudden change in temperature (internal) to reduce vasculospastic phenomenon (although evidence that spasm plays a role in the disease is questioned by some). Heat makes these patients much more comfortable and improves them temporarily.
- C Rehabilitation. Physiotherapy and psychotherapy to attempt to make the patient try to live with his disability and yet make the most of whatever assets he still retains.

## PARALYSIS AGITANS (Parkinsonism) (code No 946 4 953)

A syndrome characterized by rhythmical pill-rolling tremor of resting muscles with associated apathy and rigidity, a stooped posture, mask-like face and a peculiar gait. In later life it is usually associated with arteriosclerotic changes in the basal ganglia. In younger life it is usually associated with postencephalitic changes in the basal ganglia.

Treatment

Treatment is mainly symptomatic. Little can be done to arrest the progressive postencephalitic or arteriosclerotic changes that occur.

- A Specific Measures. A number of drugs have been found to be effective in alleviating the symptoms of Parkinsonism. These drugs are usually used in combination to obtain the optimal therapeutic result.

Cautions. In patients with paralysis agitans never stop one drug abruptly when instituting the therapy with a new one. Always introduce the new drug gradually increasing quantities while gradually reducing the old.

- 1 Artane (The pyridyl)<sup>®</sup>. Effective for sustained control of rigidity, minor tremor and akinesia. Dosage: 1 or 2 mg (40-120 gr) to 5 mg (127 gr) i.d. For ocular dyskinesias 10 mg (40 gr) i.d. Principal side reactions: none for tropic but cardiovascular side effects are minimal. Use with caution in glaucoma.
- 2 Well-down (ethanol)

## ANTISPASMODIC DRUGS

Drug	Effect On			
	Tremor	Rigidity and Spasms	Akinesia (w akn ss)	Cholergic Crisis
Artane®		X	X	X
Atropine and Belladonna Alkaloid		X		
Benadryl®	X			
Cogentin® (MK 02)	X	X		
Dexedrine®			X	
Hyoscin®	X			X
Pagitane®		X	X	X
Parparin®		X		
Paraloid® (Lysothane®)	X	X		
Rabellon®		X	X	
Stramonium	X			

(Modified from L. J. Doshay Clinical Appraisal of Parkinson Treatment The Merck Report April 1954 Reproduced with permission )

- a Atropine Solution of U S P B P (2%) Effective for spasms and rigidity Start with 3 drop doses at intervals of about 6 hours increase dosage by 1 drop every 3 days until a dosage of 10 drops t i d is reached Limited to younger patients because of danger of glaucoma in old riv Early toxic symptoms are blurring of vision dryn ss of mouth vertigo and tachycardia Excessive dosage may produce vomiting dizziness mental confusion and hallucinations
- b Belladonna Tincture U S P B P Has same effect as atropine Start with 10 drops t i d and increase gradually to 30 drops t i d Chief side effects are sam as for atropine Do not give to patients with glaucoma
- 3 Diphhydramine Hydro chloride U S P (Benadryl®) Antihistaminic for control of tremor Dosage 30 mg (3/4 gr) b i d to q i d
- 4 Cogentin® (MK 02) Most effective agent against rigidity and spasms also against tremor Excellent when combined with Artane® as well as Pagitane® or Dexedrine® Chl side reaction dryn ss of mouth Dosage Begin with 0.5 mg one or two times daily and increase as needed (up to 5 mg daily)
- 5 Dexedrine® 8 mg (1/12 gr) morning or noon or Benzedrine® 10 mg (1/8 gr) to counteract fatigue somnolence lethargy
- 6 Hyoscin Hydrobromid U S P B P Useful in control of tremors Dosage ranges from 0.3 mg (1/200 gr) b i d to q i d in elderly patients to 0.6 mg (1/100 gr) b i d to t i d in the young Distressing side effects may include somnolence dry mouth, blurred vision and drowsiness
- 7 Pagitane® Action similar to Artane® but less drying effect Useful when effect from Artane® wears off Dosage 1.25

- mg to 5 mg t i d to q i d. Contr. Indl. stated in glaucoma.
8. Carbamiphen Hydrochloride (Pariparal®) Useful in young patients as m. scler. laxant. ■ sag. f. r. ad. 50-100 mg q i d.
9. Paralid® (Lysoval®) Tablet contain 50 mg (3/4 gr) ■ y be g v n q i d in dosage ranging from 1/2 to 1 tablet.
10. Rabilon® (hyoscine hydrobromide atropine sulfate and eopolamine hydrobromide) Has relatively little effect on tremor. Tabl. ■ contain 0.5 mg (1/20 gr) of mixed belladonna alkaloids. Cl. n. in 1/4 1/2 = full tabl. t. dos. g. b. i. d. t. q. i. d. dep. on ag. and tol. ran. e. of patient. Sid. reactions: dryness of mouth and blur. d. vision. Contra. Indl. at d. in gl. om.
11. Tincture of St. monium, B. P. Especially good for control of motor tet. ion. nde. it m. nt. Dos. g. e. t. ts with 15 drops t i d and increase s. slowly to about 60 drop t i d.

### B. C. L. M. s. r. e.

1. Physiotherapy. Should include massage, stretching of muscle and active exercise when possible. Patient should be taught to exercise daily the muscles most severely affected, especially those of the hands, fingers, wrists, elbows, knees and neck.
2. Re-uranc of control of symptoms and psychological support will be g. at fully. l. ed by patient.
3. Avoid barbiturates. Permit moderate use of alcohol some times at l. t. s. on.

### P. gnosis

Prognosis is slowly progressive but it is not fatal.

## CEREBRAL VASCULAR ACCIDENTS

Cerebral vascular accidents due either to thrombosis or hemorrhage or embolism. The differential diagnosis is important in order to treat the underlying cause (see table below).

Differential Diagnosis of Cerebral Vascular Accidents

	Hemorrhage (94-95)	Thrombosis (94-95)	Embolism (94-95)
Age	45-65 yr.	Over 45 yr.	All ages
Usual and long course	Typical	Art. occlusion	C. d. disease
Onset	Gradual	Sudden or rapidly progressive	Sudden
History	Ant.	Slight or b. i.	V. b.
Motor signs	Deposits	Homolateral	Contralateral
Motor signs	Complete hemiplegia	Slight to complete hemiplegia	Slight to complete hemiplegia
Sensorial signs	Slight	Normal or slight	Variable
Headache	Usually	Usually b. i.	Ab. n. o. slight
Prognosis	Good	Good	Good

TreatmentA Acute Phase or Onset

- 1 Complete bed rest
- 2 Nursing care Handle patient carefully to avoid injury to patient and paralyzed extremities
- 3 Sedation If patient is agitated sedatives are necessary. However patients with thrombosis should not be depressed too much with sedatives
  - a Oral paraldehyde 4 cc (1 dr) in milk, fruit juice or whisky repeated as necessary
  - b Rectal paraldehyde 8-15 cc (1/2-4 dr) in 30 cc of oil
  - c I M paraldehyde 4-8 cc (1-2 dr) deep into the buttocks
- 4 Feedings If patient is unconscious or unable to swallow do not attempt to give feedings by mouth. Maintain nutrition with tube feedings or by parenteral means
- 5 Phlebotomy If hemorrhage has occurred and blood pressure is elevated phlebotomy of 500 cc may be used to reduce chances of further bleeding
- 6 Lumbar puncture If hemorrhage has occurred perform lumbar puncture very cautiously removing just enough fluid to relieve severe headache. Do not perform Queckenstedt's test in patients with suspected hemorrhage
- 7 Voiding Catheterization may be necessary if spontaneous voiding does not occur
- 8 Procaine block of the stellate ganglion and 100% oxygen inhalations have been recommended for cases due to thrombosis. This procedure may also be useful in cases of cerebral embolism but it is contraindicated in cases of hemorrhage

B State of Recovery and Convalescence The rehabilitation of the patient with hemiplegia due to cerebral vascular accident should begin early and should be intensive. Although it varies with different patients and the details are important the following phases may be delineated:

- a Bed phase
- b Standing phase
- c Stair climbing phase
- d Cane walking phase

(For details of rehabilitation program see p 547)

Prognosis

If the patient survives the acute attack the prognosis for life is good. With active rehabilitation most patients will be able to walk and care for themselves. Return of useful function to the upper extremity is rare. (These patients can be trained to achieve a remarkable degree of recovery if given adequate care and rehabilitation.) Prognosis for functional recovery is poor in those patients with severe organic mental syndrome or sensory aphasia.

HEPATO-LENTICULAR DEGENERATION (Wilson's Disease)

This extrapyramidal disease characterized by progressive intention tremor, athetosis, rigidity, dysphagia, contractures

muscle weakness, dementia, Kayser-Fleischer ring, emaciation, and associated liver disease has been reported to be due to a defect of copper metabolism.

### Treatment

Dimercaprol U.S.P. (BAL<sup>®</sup>) has been reported to be effective in removing the excessive copper. The clinically useful dose is 2.5 mg (1/24 gr)/Kg body weight by injection b.i.d. for 14-18 day periods.

## THE CONVULSIVE DISORDERS

### EPILEPSY (Idiopathic) (code No. 934)

Epilepsy is a symptom complex which may be characterized by one or more of the following manifestations (Lennox):

1. Impairment of consciousness
2. Involuntary movements of muscles
3. Disturbance of the autonomic nervous system

### Diagnosis

There are three major clinical types. The differential diagnosis is very important because the therapy of each differs. Individual may have more than one type of seizure. Electroencephalographic study is indicated in all epileptic patients.

**A. Grand Mal (code No. 930.01)** (Rule out other causes of convulsive seizure). This type occurs in all age groups. The usual form has general tonic and clonic convulsions which may begin focally and remain so or may spread without loss of consciousness (Jacksonian). They may occur in single attacks varying in occurrence from hours to years.

**B. Petit Mal (code No. 930.07)**. The usual form is characterized by a tonic type of consciousness of 3 to 30 seconds and generally a convulsive seizure occurs. During the attack there is commonly a rhythmic clonus and blinking of the eyes. It occurs most frequently in children and is rare after age 30.

**C. Psychomotor Seizure (Epileptic equivalent) (code No. 930.0)**. This form is frequent in adults and may be characterized by periods of automatic behavior. The patient is emotionally content. It usually is entirely altered from normal during the attack. The attacks vary in character and the patients are often dangerous to themselves and society.

**D. Status Epilepticus (code No. 930.806)**. Repeatedly recurring attacks of grand mal type which exhaust patient and may be fatal.

### Treatment

Epileptic in status epilepticus no treatment is given during an attack except to keep patient from being injured (e.g. biting his tongue).

**A. C and M I.** Never withdraw an anticonvulsant drug suddenly.

1. Diphenhydantoin Sodium, U.S.P. Phenytoin Sodium B.M. (Dilantin<sup>®</sup>) is the drug of choice. Give 0.1 Gm (1 1/2 gr) after

evening meal for 3 to 7 days increasing dosage by 0.1 Gm ( $1\frac{1}{2}$  gr) daily every week until seizures are brought under control. If attacks are severe and frequent may begin with 0.3 Gm ( $4\frac{1}{2}$  gr) daily on first visit. Average dose 0.4 to 0.6 Gm (6 to 9 gr) per day. After convulsive seizures are controlled the Dilantin® may be reduced if desired but should symptoms again appear the dosage should immediately be raised again.

There are a few toxic reactions to Dilantin® but most troublesome is gum hypertrophy. This is best controlled with careful mouth hygiene and gum massage. When large doses are given ataxia or drowsiness may appear (see page 354).

- 2 Phenobarbital U.S.P. Phenobarbitone B.P. If patient is on maximum dosage of Dilantin® and there is inadequate response give phenobarbital in addition in same manner and dosage as Dilantin® increasing dosage as with Dilantin® while maintaining patient at full dosage of Dilantin®.
- 3 Methylphenylethylhydantoin (Mesantoin®) If excessive gum hypertrophy results from the use of Dilantin® methylphenylethylhydantoin may be tried in its place. The dosage is the same. Mesantoin® may be effective where grand mal and petit mal coexist. Do not suddenly change to Mesantoin® but gradually substitute for Dilantin®. Combinations of both may prove more useful than the individual drugs. When using Mesantoin® special precaution should be observed for toxicity (see page 354).

In the event of failure of the above drugs bromides Phenurone® Mysoline® Mebaral® or Hibicon® should be tried (see chart page 353).

## II F (It Mal)

- 1 Very mild state. If attacks are infrequent (less than 1 per day) give no treatment or treat only with small doses of phenobarbital.
- 2 Mild state
  - a Amphetamine sulfate 5 to 10 mg ( $1\frac{1}{2}$  to 2 gr) 2 to 3 times daily may be attempted. Do not use amphetamine if patient also suffers with grand mal because use this may precipitate grand mal attacks.
  - b Glutamic acid 8 to 10 Gm (2 to 2½ dr) daily may decrease the number of attacks.
- 3 Moderate and severe states
  - a Trimethadione U.S.P. (Tridione®) is the drug of choice. Trimethadione is very effective in petit mal epilepsy but unfortunately is not an entirely safe drug since it causes bone marrow depression in some individuals. Therefore this drug is used perform CBC once or twice a week for the first month then every two weeks for two or three months and monthly thereafter. Dosage. Begin with 0.3 Gm (5 gr) daily and increase the daily dose by 0.3 Gm (5 gr) every 7 days until attacks are controlled. Do not give more than 2 Gm (30 gr) daily.
  - b If grand mal seizures occur also trimethadione may

aggravate this tendency the frequency it may be necessary to administer medication for 2 and mal sei simultaneously and in some cases stop the trimethadione. P ram thiadione (P adione®) has recently been developed. It is said to be less toxic than trimethadione. It is almost equally effective in petit mal attacks and may be effective where other drugs fail. The same precautions as for trimethadione must be observed (see page 354). Milontin® Phenuron® phenobarbital P enderol® Mebaral® or a ketogenic diet may prove useful where the above drugs fail.

**C P y homoto Epil p y** Patients must be watched and guarded to prevent injury to themselves or others.

1 Diphenylhydantoin Sodium USP Phenytoin Sodium BP (Dilantin®) with or without phenobarbital phenobarbital employed as first and mal epilepsy treatment if choice.

2 Phenylacetyl Phen mid N N R (Ph one®) has proved to be effective in control of psychomotor epilepsy. The drug is administered initially as 0.5 Gm (7½ gr) 3 times daily and increased until symptoms are controlled. Up to 3.0 Gm (75 gr) daily divided into 3 to 5 equal doses. The drug is very toxic and precautions must be observed with its use (see page 354).

**D St Epil pil**

1 P aid hyd 1 2 c (½ ½ dr) in 3 times the volume of saline solution 1 V slowly if convulsion does not stop repeat dose immediately. Glucose 12 cc (2 3 d) 1 M.

2 Amyl Sodium® phenobarbital sodium 0.5 1.0 Gm (1½ 15 gr) 1 V may be given.

3 Chloral hydrate may be administered if all other measures fail.

4 Dilantin® As soon as edatim is seen as effective. As at maintenance high dose while patient is still unconscious 0.5 Gm (1½ gr) Dilantin® dissolved in water 10 doses until seizure is under control (maximum 10 doses).

**E D tion f T** In the M t pil patient must receive therapy for life. However, if seizure occurs, immediately continued for 3 to 5 years. If convulsion drug may be slowly (over 12 years) withdrawn. If seizure occurs, immediately occur.

**F Gr 1 M su**  
1 A quantity of 1 with his diet. In case of this may be accomplished by re-diagnosing about the disease (see page 356).  
2 A diet of rice or up to 100 and driving.  
3 Avoid inactivity. It is important to maintain regular program of physical activity.  
4 Keep patient in optimum physical condition and avoid overexertion.

5 Forbidden alcohol.  
6 Treatment of fever when this is indicated.  
7 Instruction of patient in behavior necessary for first aid.  
8 An individual should be advised at all times.



## DRUGS USED IN EPILEPSY

Dose	Indication	Adult Dosage	Therapeutic Effects	Precautions	Remarks
Phenyhydantoin (Dilantin) (Phenylhydantoin)	Grand mal and petit mal	0.3-0.5 Gm (5-7 1/2 gr) in divided doses	Phenylhydantoin 1. Good type of tonic 2. Nausea 3. Rash 4. Itching	1. Mildly good of tonic 2. Reduce if too itchy 3. Stop if too itchy	Not drug of choice for grand mal and petit mal
Sodium Valproate (Miltal) (Miltal)	Grand mal and petit mal	0.3-0.5 Gm (5-7 1/2 gr) in divided doses	1. Nausea 2. Itching 3. Rash 4. Itching	1. Mildly good of tonic 2. Reduce if too itchy 3. Stop if too itchy	Not drug of choice for grand mal and petit mal
Trimethadione (Glim) (Glim)	Grand mal and petit mal	0.3-0.5 Gm (5-7 1/2 gr) in divided doses	1. Nausea 2. Itching 3. Rash 4. Itching	1. Mildly good of tonic 2. Reduce if too itchy 3. Stop if too itchy	Not drug of choice for grand mal and petit mal
Phenobarbital (Luminal) (Luminal)	Grand mal and petit mal	0.3-0.5 Gm (5-7 1/2 gr) in divided doses	1. Nausea 2. Itching 3. Rash 4. Itching	1. Mildly good of tonic 2. Reduce if too itchy 3. Stop if too itchy	Not drug of choice for grand mal and petit mal
Phenytoin (Dilantin) (Dilantin)	Grand mal and petit mal	0.3-0.5 Gm (5-7 1/2 gr) in divided doses	1. Nausea 2. Itching 3. Rash 4. Itching	1. Mildly good of tonic 2. Reduce if too itchy 3. Stop if too itchy	Not drug of choice for grand mal and petit mal

[illegible]

## Identification Card

THIS PATIENT HAS EPILEPSY		
Name_____		
Address_____	Phone_____	
Put a padded stick or spoon in his mouth to protect his tongue Keep him from injuring himself		
Doctor's Name_____		
Address_____	Phone_____	

■ Education of the Epileptic Patient1 Books for the epileptic patient

- a Lennox Science and Seizures Harper and Bros
- b Putnam On Convulsive Seizures a Manual for Patients  
J B Lippincott Co

## 2 Encourage the epileptic patient to become a member of The American Epilepsy League Inc Room 403 50 State St Boston 9 Mass Patients may receive information regarding research and treatment from this organization

**DISEASES OF THE CRANIAL NERVES****TRIGEMINAL NEURALGIA (code No 964 x30)**

Trigeminal neuralgia is characterized by a sudden attack of excruciating pain of short duration over any of the distribution of the 5th cranial nerve The attack is normally precipitated by stimulation (usually mild) of a trigger zone in the area of the pain

Treatment

A Medical treatment is generally unsatisfactory but the following usually are tried before resorting to surgery

- 1 Trichloroethylene (15-20 drops per day by inhalation from handkerchiefs in single or divided doses)
- 2 Short wave diathermy over the area of exit of the nerve 1 hour daily for 6 days
- 3 Massive doses of vitamin B<sub>12</sub> (1000 micrograms daily by injection for 10 days) have been reported to relieve the severe pain of trigeminal neuralgia

B Surgery must be resorted to if there is no relief from these

**BELL'S PALSY (Peripheral Facial Paralysis) (code No 965 y10)**

A paralysis of all the muscles of one side of the face usually precipitated by exposure chill or trauma

Treatment

Assure the patient that recovery usually occurs generally in

8 weeks in many take up to 1 2 years in older patients

#### A Protection of Face

- 1 Keep face warm and avoid further exposure
- 2 Protect eyes with a patch if necessary
- 3 Avoid wind and dust

#### B Physiotherapy

- 1 Support by use of tape or wire from angle of mouth looped about the ear
- 2 Electrical stimulation may be used to help prevent atrophy of muscles. Do every 2 days after the 14th day
- 3 Gentle massage in an upward direction for 10 minutes 2 3 times a day of the involved muscles may help the tonus
- 4 Heat from infra red lamp may hasten recovery

### MENTIERS SYNDROME (code No 200)

Mentiers syndrome is a symptom complex of unknown etiology which involves the labyrinthine portion of the 8th nerve. It is manifested by sudden recurrent attacks of vertigo, nystagmus, vomiting and tinnitus and by progressive deafness

#### Treatment

A Sp. in M Non available  
B C. in M

- 1 Reassure as to importance of many of these patients have a marked psychical overlay
- 2 Salt free diet and ammonium chloride 1 2 Gm (15 30 g l) q i d may be helpful
- 3 Nitrolic acid (niacin) (not nicotinic acid) 50 100 mg (3 4 to 1 1/2 gr) 1 V b i d to t i d 100 mg (1 1/2 gr) 1 l y 5 to 6 times daily has been found useful
- 4 Thiothamizone sulphate (Dramamine) in the form of 50 100 mg (3/4 1 1/2 g) 3 or 4 times daily after meals to be of benefit to some patients

### DISORDERS OF EQUILIBRIUM

#### ACUTE LABYRINTHITIS (code No 285 910)

Acute labyrinthitis is a disorder of the inner ear characterized by a sudden onset of vertigo, nystagmus, tinnitus and hearing impairment. It is usually associated with inflammation of the labyrinthine portion of the 8th nerve.

#### Treatment

A Sp. in M Non available  
B C. in M

- 1 Bed rest for 1 2 days and avoid all symptoms
- 2 Drugs  
a. Antibiotics are of little value unless the infection is associated

- infection of middle ear or mastoid
- Antihistamine drugs may be of some value (as for motion sickness see below)
- c Sedation is generally helpful Phenobarbital phenobarbital 15 60 mg ( $\frac{1}{4}$  1 gr ) t i d to q i d

### MOTION SICKNESS (code No 010 576)

Motion sickness is an acute illness characterized by anorexia nausea dizziness and vomiting Many factors play a role in its production the principal ones being visual kinesthetic and psychological Physiologically the vestibular apparatus appears to be involved

#### Prophylactic Treatment is of M at Importance

- A The antihistaminics appear to be of benefit Dimenhydrinate (Dramamine®) or diphenhydramine hydrochloride (Benadryl®) given in doses of 50 100 mg ( $\frac{3}{4}$  1½ gr ) q i d is stated to be very effective
- B Parachloramine (Donamine®) is a long acting effective agent Usual dose 50 mg ( $\frac{3}{4}$  gr ) every 6 h hours
- C Parasympathetic depressants alone or in combination with mild sedatives Scopolamine hydrobromide or atropine sulfate 0.2 0.4 mg ( $\frac{1}{300}$   $\frac{1}{150}$  g ) every 3 to 6 hours
- Mild Sedation Phenobarbital phenobarbital 15 30 mg ( $\frac{1}{4}$   $\frac{1}{2}$  gr ) every 3 to 6 hours may help prevent attacks

### PERIPHERAL NEURITIS (code No ■ y10)

Peripheral neuritis can be caused by a large number of factors both local and general There may be either sensory involvement (with pain paresthesias and other subjective sensory disturbances) or motor involvement (weakness and paralysis) but more frequently both

- A Toxic Form E g lead arsenic mercury or diphtheria toxins
- Infections Guillain Barre type of multiple neuritis
- C Deficiency Type Especially of the B complex (beriberi) is often associated with superimposed toxic neuritis such as the alcoholic polyneuritides diabetic neuropathy
- Traumatic Due to direct injury to the nerve

#### T estime 1.

Treatment in each depends upon the etiological factors

#### A Specific Treatment

- 1 Remove noxious agent e g alcohol lead source
- 2 Vitamin B complex Attempt to obtain optimal metabolism of nerve tissue by liberal use of vitamins especially B complex Thiamine hydrochloride aneurine hydrochloride 15 mg ( $\frac{1}{4}$  gr ) t i d to b i d orally or parenterally and dried yeast (brewer's yeast) 10 30 Gm ( $\frac{1}{10}$  1 oz ) daily f r entire B complex (see B vitamins pag s 61 and 62)

#### B General Treatment

- 1 Bed rest Place patient in bed if possible and avoid use of affected limb If lower extremity affected keep extremity at foot of bed to prevent pressure of bed covers
- 2 Analgesics necessary to control pain (see page 38)
- 3 Physical therapy (see page 330)
  - a After pain has subsided physical therapy (massage and passive motion) may be of value Encourage active motion at same time
  - b Prevent contracture by means of splints and passive stretching

## HERNATION OF INTERVERTEBRAL DISK (code No 2511 9x9)

Compression and injury to nerve root may be caused by herniation of an intervertebral disk Most commonly the lumbosacral intervertebral disks (L5 S1 or L4 L5) are affected in these cases the symptom complex of low back pain impaired range of motion of lower back paravertebral lumbar muscle spasm and pain radiating along the sciatic distribution are commonly encountered Onset of clinical complaints is frequently related to period involving low back strain back injury or falls The initial period may be followed by an interval of symptomatic improvement

Exaggeration of symptomatic complaints follows frequently upon laughing training no lifting

Tenderness in sciatic notch along course of the sciatic nerve impaired straight leg raising diminished ankle jerk impaired sensation over distribution of L5 or S1 may be demonstrated

Characteristic roentgenological defect in the sacrocaudal space is usually produced by a herniated intervertebral disk and is easily demonstrable by myelography

Treatment

A General Management

- 1 Encourage patient to rest heat applied locally to back and leg
- 2 Use of a bed board under mattress as indicated
- 3 Traction to the lower extremity is frequently beneficial
- 4 The avoidance of excessive physical effort and strain is essential to minimize recurrence of symptoms after the initial period
- 5 Use of low back belt brace or supports may be beneficial It is important to instruct patient as to proper method of bending lifting (with knees flexed) and carrying (with object held close to body)

B Surgical Management

When the response to conservative measures is poor when the current disability of the patient is great the surgery is indicated. Giving relief of the major complaints of most patients is pain usually follows the successful removal of the offending herniated disk Recovery of other neurological functions (impaired motor power the atrophy skin sensory changes) may be expected later

## MYOPATHIES

### MYOTONIA

(Congenital code No 270 044) (Acquired code No 270 x20)

*Myotonia is a disorder characterized by difficulty in relaxation of skeletal muscles following contraction which is initiated either by voluntary effort or by mechanical or electrical stimulation. It is important to differentiate this disease from myasthenia (see below) because treatment with neostigmine or potassium aggravates myotonia.*

#### Treatment

Quinine sulfate 0.306 Gm (59 gr) 2-4 times daily may give dramatic relief from symptoms.

### PROGRESSIVE MUSCULAR DYSTROPHY (code No 270 9x9)

*A disorder characterized by progressive wasting and weakness of muscles with associated pseudohypertrophy (fat infiltration) of certain muscle groups. It is important to differentiate this from myasthenia gravis because the latter can be benefited by treatment.*

#### Treatment

None of value. It has been suggested that inability to metabolize vitamin E may play a role in the disease but parenteral or oral administration of this substance has been of no benefit.

### MYASTHENIA GRAVIS (code No 270 562)

*Myasthenia gravis is a disorder characterized by weakness and marked fatigability of voluntary muscles. Recovery from weakness or fatigue occurs with rest or specific medications. The disease progresses by natural or spontaneous remissions and relapses. A therapeutic test of 1.5 mg (1/45 gr) neostigmine methylsulfate with 0.6 mg (1/100 gr) atropine sulfate (diagnostic ampule) subcut may be used. This causes relief of symptoms.*

#### Treatment

**A. Emergency Treatment.** Patients may suddenly develop inability to swallow or respiratory crises. Patient should always carry 2 ampules of 1 mg (1/120 gr) of Neostigmine Methylsulfate U.S.P. This should be given immediately subcut or I.M. if severe symptoms develop. The patient should be placed under medical care at once and if additional neostigmine is needed 1 mg (1/120 gr) may be given parenterally 3 times in an hour until adequate response is obtained.

In spite of administration of increasingly large amounts of neostigmin progressive weakness of muscles of respiration may occur which may be fatal in some cases.

1. When such an event can be anticipated tracheotomy and oxygen equipment suction apparatus and respirator should be available.

- 2 Following a heotomy patient is placed in respirator  
Oxygen administered as needed. Neostigmine withheld  
t open 0.6 mg (1/100 g) administered to keep airway  
dry and suction if airway employed
  - 3 Fluid and electrolyte balance is maintained during initial  
respiration period
  - 4 Antibiotics are given to prevent pneumonia
  - 5 After a few days it is usually possible to gradually decrease  
the amount of neostigmine tolerated
  - 6 In patients who are violent or restless, sedation may occur in  
me that necessitates frequent administration of sedatives
- B Specific Measures**
- 1 Neostigmine Bromide USP BP 15 mg (1/8 gr) oral  
tablets. Dose varies from 4 to 12 tablets daily. Begin with  
1 tablet every 4 hours (4 times a day) and increase dose  
as required to give relief
  - 2 Ephedrine sulfate 12 mg (1/8 gr) with each dose of neostigmine  
often enhances the action of neostigmine
  - 3 Potassium has also been found of value to supplement neostigmine. It must however be given in 15 to 20 times a day. This  
is contraindicated if cardiac arrhythmias are present
  - 4 Although a more favorable result has been reported with the  
use of corticotropin (ACTH) or cortisone therapy so vari-  
able that the outcome of these drugs cannot be considered  
Some patients have been made weakly temporarily
  - 5 Tension may rise to 20 to 30 mm Hg and 25 to 50 mg of M g l s  
given orally in 20 to 30 seconds
- C Complications**
- 1 A patient is not with his disease using simple terms
  - 2 Patient in good nutrition and health
  - 3 Thymectomy is indicated to benefit some patients
- D Prognosis**
- Prognosis is usually good and a diagnosis and method of treatment

### THIS PATIENT HAS MYASTHENIA GRAVIS

Name \_\_\_\_\_ Phone \_\_\_\_\_  
Address \_\_\_\_\_

If observed to be behaving strangely or if he is found  
unconscious call for physician or ambulance

Physician Name \_\_\_\_\_  
Physician's Address \_\_\_\_\_ Phone \_\_\_\_\_

Two ampules of neostigmine and a syringe are needed  
re in his possession. These must be administered  
hypodermically in the upper arm innervated by



Management During Pregnancy

Immediately after delivery children of patients with myasthenia may have severe signs of the disease. Immediate treatment with neostigmine is necessary to preserve life. After a few days the symptoms may disappear and the child thereafter does not suffer from myasthenia.

**FAMILIAL PERIODIC PARALYSIS (code No 270 x95)**

A disease of unknown etiology characterized by recurrent attacks of flaccid paralysis of the muscles of the trunk and extremities and by a lowering of the serum potassium level during the attack. Immediate relief of symptoms by administration of potassium chloride is usually diagnostic.

Treatment

- A Potassium chloride 10 Gm (75-150 gr) orally when diagnosis has been made and then 5 Gm (75 gr) b i d q i d during acute episode as needed to prevent weakness or paralysis.
- B In emergencies only may give 1 Gm (15 gr) potassium chloride in 50-80 cc (2 oz) distilled water injected very slowly i v. This is a dangerous procedure (see page 25).

Prophylaxis

Potassium chloride 5 Gm (75 gr) at bedtime is advised by some to prevent attacks.

## Chapter 14

# METABOLIC AND ENDOCRINE DISEASES

## DISEASES OF THE PITUITARY

In the diagnosis and treatment of endocrine disorders it must be remembered that there is a very close interrelationship of the various endocrine organs. Not only do hormones exert a profound effect on all tissues of the body but the endocrine glands also exert strong influences upon one another. For this reason the manifestations of endocrine disease may be either primary (a given endocrine gland or a secondary due to involvement of target glands. For example the patient with hypopituitarism may present with a picture of frank myxedema and unless it is appreciated that the primary disturbance is in the pituitary administration of thyroid hormone without simultaneous use of corticosteroids is a very serious addition to crisis.

### PANHYPOPITUITARISM (code No 841 TTT) HYPOPITUITARY CACHEXIA (Simmonds Disease) (code No 841 TTT)

Organic hypopituitarism is due to destruction of the anterior pituitary which may be caused by tumors of the gland or postpartum hemorrhage (Sheehan's syndrome).

The term panhypopituitarism is possibly a misnomer since the significant variation in symptomatology from case to case may be due to varying degrees of deficiency of the several anterior pituitary hormones. Hypopituitary cachexia is also a misleading term since these patients may be of normal weight or may actually be obese. Symptoms of organic hypopituitarism usually include weight loss, weakness, sensitivity to cold, loss of appetite and in the males amenorrhea. Physical examination reveals loss of axillary and pubic hair and atrophy of skin and genitalia. Laboratory findings are as follows: low B.M.R., low radiiodine uptake, or PBI, decreased insulin sensitivity, low urinary 17-ketosteroid excretion and low urinary gonadotropin excretion. These manifestations are due largely to lack of pituitary stimulation of the various endocrine glands (thyroid, gonads and adrenal).

Differentiation of panhypopituitarism from anorexia nervosa (functional hypopituitarism) may be difficult. Psychic disturbances may be found in both conditions although a history of specific emotional stress or long standing psychiatric symptoms is more suggestive of anorexia nervosa. The nervosa patient is usually more alert and active and more able to withstand stress. The axillary hair is usually not lost in anorexia nervosa but is almost always lost in organic hypopituitarism. The low urinary gonadotropin level (less than 3 mouse units per 24 hrs.) of hypopituitarism may be of aid in diagnosis but is not definitive. Improvement following special feeding technique and failure to respond to specific endocrine treatment would further suggest anorexia nervosa.

### Treatment.

There is no effective pituitary replacement preparation. Therapy must therefore be aimed at correcting the end organ deficiencies. This must be continued throughout life. Almost complete replacement therapy can be carried out with cortisone.

A Cortisone or Hydrocortisone 7.5 to 25 mg ( $\frac{3}{8}$  to  $\frac{1}{2}$  gr.) per day is usually adequate. This should be given in divided doses 3-4 times daily.

B Thyroid Thyroid (and insulin) should rarely if ever be used in panhypopituitarism unless the patient is receiving cortisone. Because of lack of adrenal cortical function patients are exceedingly sensitive to these drugs. For this reason one should exercise special care in differentiating myxedema from hypopituitarism often a difficult problem.

Begin with small doses of 15 to 30 mg ( $\frac{1}{4}$  to  $\frac{1}{2}$  gr.) daily and gradually increase to tolerance. 60 to 100 mg (1 to  $1\frac{1}{2}$  gr.) is usually adequate.

### C Sex Hormones

1 Testosterone May be used in both males and females primarily for its tissue building (protein anabolic) effect. Dosage: Testosterone Propionate U.S.P. B.P. 25 mg ( $\frac{3}{8}$  gr.) daily 3 times weekly I.M. or Methyl testosterone U.S.P. B.P. 10 to 20 mg ( $\frac{1}{6}$  to  $\frac{1}{3}$  gr.) orally in males. In females the dose of these drugs is half that for males. If virilizing signs appear in the female the drug should be stopped and these signs will disappear. Virilizing signs usually do not result if the dose is kept under 400 mg. per month.

2 Estrogens These agents are useful in the female for their mild anabolic effect, their effect on secondary sex characteristics and their possible neutralizing effect on androgens. Diethylstilbestrol U.S.P. Stilboestrol B.P. 0.3 to 0.6 mg ( $\frac{1}{210}$  to  $\frac{1}{100}$  gr.) or Ethinyl Estradiol N.N.R. 0.02 to 0.05 mg daily orally.

Note Sex hormones especially estrogens should be employed cautiously in young panhypopituitary patients or the epiphyses will close before maximum growth is achieved.

**Pituitary gigantism** which is caused by adenoma or cancer of the pituitary is principally a result of hypersecretion of the growth factor. Growth which occurs primarily at the epiphyses of long bones is symmetrical and generalized and patients may attain a stature of seven feet or higher. Growth is possible only if there is oversecretion of hormone occurs prior to the onset of epiphyseal closure. Local pressure symptoms of the pituitary tumor may cause headaches, dizziness and visual disturbances. An elevated serum inorganic phosphorus is one of the biochemical signs of activity. Glycosuria may be present.

#### Treatment

Treatment is aimed at suppressing the pituitary growth hormone. If gigantism is found in adolescence and there is little or no encroachment of the tumor upon adjacent structures (i.e., optic nerves), give Testosterone Propionate.

**A. Endocrine Therapy.** U.S.P. B.P. 25-50 mg ( $3\frac{3}{4}$  gr) 1 M daily and give estrogen. Ethinyl Estradiol 1 N.N.R. 0.1-0.5 mg by mouth daily. Males should receive 100 mg of testosterone and females 10 mg of estrogen. This therapy will prevent further growth by causing closure of the epiphyses and may inhibit pituitary growth hormone secretion. Do not use methyl testosterone.

**B. Surgery and X-ray Therapy.** If there is marked encroachment upon visual fields, x-ray therapy appears to be of value. If x-ray therapy does not control tumor growth, surgery must be considered.

#### ACROMEGALY (code No 841 7762)

Hypersecretion of the growth factor of the anterior pituitary due to adenoma or carcinoma of the gland which develops after the bony epiphyses have fused results in a clinical picture of progressive growth of soft tissues and thickening of bone. The disease usually has its onset during the 2nd or 3rd decades. It is characterized by conspicuous enlargement of the jaw, nose, supraorbital ridges, hands and feet and thickening of the skin and visceral enlargement. Elevation of the inorganic serum phosphorus is an important diagnostic point. If the serum phosphorus is normal, the disease is probably inactive and requires no further treatment.

#### Treatment

**A. Pituitary Gigantism (see above).** Favorable responses have been reported in some cases with endocrine therapy.

## DIABETES INSIPIDUS (Due to Unknown Cause code No 842 779)

Destruction of the posterior pituitary or impaired function of the supraoptic nuclei or of tracts from these nuclei to the posterior pituitary (63% of cases being due to tumor) *causes* the condition known as diabetes insipidus. This is manifested by severe thirst and marked polyuria. A polyuria of over 5 liters per day with specific gravity below 1.006 is highly suggestive of diabetes insipidus. The diagnosis is established by the Hickey Hare test. This test consists of (1) I V infusion of hypertonic salt solution which in patients with diabetes insipidus causes an increased urine flow and (2) administration of a test dose (0.2-0.3 cc) Vasopressin Injection U.S.P. (Pitressin®) which causes a decrease in urine flow.

### Treatment

- A Specific Therapy. Vasopressin Tannate M.N.R. (Pitressin Tannate<sup>4</sup>) 1 cc in oil I.N. is the treatment of choice. It is effective for from 24 to 72 hours. It is usually best to administer the drug in the evening so that maximal results can be obtained during sleep. Patients learn to administer the drug themselves and the dosage is adjusted as necessary. Posterior pituitary snuff inhaled 2-3 times a day may be used but it is quite irritating and absorption is uncertain. The dose varies from 30-60 mg (1/4-1 gr). The aqueous preparation (Vasopressin Injection U.S.P. Pitressin®) is rarely used in chronic treatment because of its short duration of action (1-4 hours).
- B Non specific Measures. Mild cases (or Pitressin® resistant cases) require no treatment other than adequate fluid intake.
- C X ray therapy may be used in treatment of some cases of tumor (e.g. eosinophilic granuloma).

## THYROID

The thyroid gland utilizes inorganic iodine to form a complex physiologically active thyroxine protein compound that is necessary for normal function of the organism. The normally functioning gland removes the very low concentrations of inorganic iodine present in blood synthesizes it through diiodotyrosine to thyroxine and possibly triiodothyronine and liberates the active materials probably a combination of the 2 or 3 organic compounds above or derivatives thereof bound to protein. When an excess of inorganic iodine is present in the blood the thyroid cells pick it up and store it in organic form in the colloid of the follicle. Under the influence of the anterior pituitary this colloid material can be released with its active principle into the blood stream. The circulating organic iodine is quite constant in health ranging from 4-8 micrograms per cc of blood.

The requirements for iodine are very slight and difficult to estimate. About 20-200 (0.02-0.2 mg) micrograms per day are

probably necessary. At times of stress especially in puberty and during pregnancy and lactation the requirement is rise as high as 200-1000 micrograms (0.2-1.0 mg) daily.

### Abnormal Metabolism

Although the iodine requirements are very slight in many areas of the United States and elsewhere the requirements cannot be met from local food and water sources.

**A Simple Iodine Lack (Simple Goiter)** Endemic goiter or colloid goiter is characterized by enlargement of the thyroid gland and is due to relative or absolute iodine deficiency. There is often a history of living in an iodine-deficient geographic area (endemic goiter areas). Symptoms appear only if the enlargement is sufficient to cause pressure on surrounding structures (esophagus, trachea or recurrent laryngeal nerve). There is no evidence of either hyper- or hypofunction and accordingly the B.M.R., serum protein bound iodine, cholesterol and radioiodine ( $^{131}$ I) uptake are usually normal.

**B Hypothyroidism** In this condition the gland fails to manufacture adequate hormone. This may have various causes: (1) more complete iodine lack than in simple goiter; (2) inflammatory destruction of the gland (thyroiditis); (3) excessive surgical removal; (4) failure of the pituitary to elaborate thyrotropic pituitary. In hypothyroidism the B.M.R., radioiodine ( $^{131}$ I) uptake and blood organic iodine are frequently low (the latter is below 4 micrograms per cent).

**C Hyperthyroidism** This disease is characterized by excessive secretion of thyroid hormone. The causes of this are obscure but it is believed that in many cases the primary difficulty may be excessive secretion of antipituitary thyrotropic hormone. This excessive secretion causes a speeding up of metabolic functions especially the oxidation mechanism of cells. There is a resulting increase of B.M.R. the blood levels of organic iodine are frequently above 8 micrograms per cent and the  $^{131}$ I uptake is high.

## DISEASES OF THE THYROID

### NON TOXIC DIFFUSE GOITER (code No. 810.943) (Simple Goiter)

#### Diagnosis

There is often a history of living in an iodine-deficient area. Symptoms appear only if the enlargement is so great as to cause pressure on surrounding structures (esophagus, trachea or recurrent laryngeal nerve). The B.M.R. and the serum protein bound iodine and radioiodine ( $^{131}$ I) uptakes are normal.

#### Treatment

##### **A Specific Measures**

Iodine therapy (exactly). If the enlargement is discovered early it may disappear completely with adequate iodine. Five drops daily of saturated solution of potassium iodide or

Strong Iodine Solution U S P Aqueous Solution of Iodine B P (Lugol's solution) in  $\frac{1}{2}$  glass water is adequate therapy. Continue therapy until gland returns to normal size then keep on maintenance dosage or use iodized table salt.

- 2 Iodine therapy (late) If the enlargement is of long standing iodine therapy as above may be used but much regression in the size of the gland should not be expected.
- 3 Some clinicians use Thyroid U S P B P 50-120 mg (1-2 gr) in these patients especially if the goiter is multi-nodular.

#### Indications For Surgery

- 1 Signs of pressure If signs of pressure are present the gland should be removed surgically.
- 2 Potential malignancy Surgery should be considered for any thyroid gland with a single nodule for the chances of a single nodule being malignant are quite high. This is particularly true in younger people and when there is no response to treatment.

#### Prophylaxis

With an intake of 100-200 micrograms of iodine daily this condition should not occur. During times of stress (puberty, pregnancy and lactation) the upper limits of this dose may prove necessary. This amount is satisfied by 1-2 Gm (15-30 gr) of iodized salt (1:5000-1:10,000 parts iodine) daily.

### HYPOTHYROIDISM (code No 610 7722)

#### Diagnosis

A Symptoms Early weakness, easy fatigability, cold sensitivity.

B Signs

- 1 Early These are few and may be difficult to find. Dry skin and hair, brittle nails, and menstrual disturbances are suggestive.
- 2 Later Hair tends to fall out (especially eyebrows), sweating diminishes, face becomes puffy (early about the eyes), then non-pitting edema spreads to the rest of the body. Patient may develop anemia and heart disease.
- 3 Obesity is an uncommon finding in true hypothyroidism.

C Laboratory Diagnosis

- 1 Low B M R A B M R below -30% is suggestive but not diagnostic of hypothyroidism. A low B M R does not necessarily mean hypothyroidism; this is especially true in obese patients. (See Obesity p 390).
- 2 Serum iodine A low protein bound iodine of under 4 micrograms per cent.
- 3 Decreased radiiodine ( $^{131}$ ) uptake (below 10% in 24 hours).
- 4 Other significant findings include elevated blood cholesterol (above patient's normal) and in severe cases anemia.

#### Treatment

A Specific Therapy Thyroid U S P B P is the preparation of choice. Initial dosage varies with the severity of the hypothyroidism.

- 1 Patients with severe myxedema myxedema heart disease or elderly patients with hypothyroidism with other associated heart disease Begin with small doses 15 mg ( $\frac{1}{4}$  gr) daily for 1 week and increase dose every week by 15 mg ( $\frac{1}{4}$  gr) daily up to a total of 100 to 200 mg ( $1\frac{1}{2}$  to 3 gr) daily This dosage should be continued until signs of hypothyroidism have vanished or toxic symptoms appear then stabilize dosage so as to maintain the B M R of protein bound iodine at normal or just below the level of toxicity (see below under Hyperthyroidism)
- 2 Patient with early hypothyroidism may be started with larger doses 30 mg ( $\frac{1}{2}$  gr) daily increasing by 30 mg ( $\frac{1}{2}$  gr) every week in the limit of tolerance
- 3 Chronic maintenance Each patient's dose must be adjusted to obtain the optimum effect. Most patients require 100 to 150 mg (1 to 2 gr) daily for maintenance Optimum maintenance dosage can be estimated by following B M R, protein bound iodine and plasma histiocyte but clinical judgment is usually the best guide

#### D. Needle test of thyroid

- 1 Questionable diagnosis If any patient can tolerate about 200 mg (3 gr) daily of thyroid the diagnosis of hypothyroidism should be questioned Normal individuals and obese and other non-hypothyroid individuals can tolerate doses up to 300 to 500 mg ( $4\frac{1}{2}$  to  $7\frac{1}{2}$  gr) daily without changes in B M R or development of toxic symptoms
- 2 Non-specific use of thyroid The use of thyroid medication as a non-specific stimulating therapy is mentioned only to be condemned It has been shown that the doses usually employed (100 to 200 mg or  $1\frac{1}{2}$  to 3 gr daily) are ineffectual in altering the metabolism of a normal individual

### HYPERTHYROIDISM

In the past it had been customary to classify hyperthyroidism according to the gross anatomical characteristics of the gland as

- 1 Diffuse Toxic Goiter (which is associated with exophthalmos Graves's disease) (code No. 810.943.6)
- 2 Nodular Toxic Goiter (code No. 810.952.6)
- 3 Hyperthyroidism Without Goiter (code No. 810.771)

However, since treatment is aimed primarily at the disturbed physiology and since there is no evidence that there are any differences in the histopathology, this is discussed under the common heading of hyperthyroidism as follows:

#### Diagnosis

- A Symptoms Nervousness irritability, excitability and weight loss in spite of excessive appetite and food intake

#### B Signs

- 1 Patient is so quick in his movements
- 2 Warm and moist skin
- 3 Fine tremor of extremities usually present in a relaxed state
- 4 Marked weight loss and emaciation
- 5 Goiter (at times a bruit may be heard over gland)



- 6 In exophthalmic variety exophthalmos may be marked
- 7 Cardiovascular findings vary most common is tachycardia but in older patients especially with long standing hyperthyroidism cardiac failure and auricular fibrillation are not uncommon

#### ■ Laboratory Findings

- 1 Elevated B M R Elevated B M R may be present in other conditions such as fever malignancy (especially leukemia)
- 2 Elevated hormonal iodine Above 8 micrograms per cent is suggestive but this may be seen in pregnancy malignancy with excessive tissue destruction burns during iodine therapy and after therapeutic or diagnostic use of iodine containing organic compounds (e g drugs used for gall bladder or kidney visualization)
- 3 Increased  $^{131}$  uptake may be diagnostic
- 4 The blood cholesterol level may be low

#### Treatment

Treatment is aimed at stopping the excessive secretions of the thyroid Several methods are in use and the method of choice is still open to debate and varies with each case The most widely accepted method however is adequate preparation followed by subtotal surgical removal

**A Subtotal Thyroidectomy** This is probably still the method of choice since it demands the least in follow up care Adequate preparation is of the utmost importance One or 2 drugs are generally necessary for adequate preparation iodine and/or one of the thiouracil group of drugs

- 1 Iodine Has been used for years Iodine is given in daily dosages of 5-10 drops of Strong Iodine Solution U S P Aqueous Solution of Iodine B P (Lugol's solution) or saturated solution of potassium iodide with nonspecific therapy (see below) until the B M R has dropped to below 20% the signs and symptoms have decreased and the patient has begun to gain weight The disadvantages with iodine are as follows
  - a A few patients may not respond especially those who have received iodine recently
  - b If there is too long a wait before surgery the gland may escape and the patient develops a more severe hyperthyroidism than before
  - c It is generally impossible to bring the B M R to normal with iodine
- 2 Thiouracil drugs Recently several thiouracil drugs or similar derivatives have been introduced They are Propylthiouracil U S P B P Methylthiouracil N N R Methimazole N N R and one containing iodine in the molecule iodothiouracil (Itrumil®) The modes of action of the first three are probably identical that of iodothiouracil is still not entirely clear
  - a Propylthiouracil U S P B P This drug has been most widely used and appears to be the least toxic It is the thiouracil preparation of choice The mode of action of this drug is such that when given in adequate dosage

it prevents the thyroid gland from incorporating inorganic iodine into its organic (hormonal) form. This effect is very rapid (within a few hours) and continues as long as the drug is given. The gland continues to attempt to manufacture thyroid hormone (remains hyperplastic or becomes more so) but none is made. Because of this the B.M.R. invariably falls, the rate of fall depending upon the total quantity of previously manufactured protein-bound iodine available from the gland or in the circulating blood. (More protein-bound iodine is present if iodine has previously been given.) The average time required for the B.M.R. to return to normal is about 4-6 weeks. If the drug is continued the B.M.R. will continue to fall until the patient becomes myxedematous.

- (1) The drug appears to be ideal except for 2 factors
  - (a) Danger of toxic reactions, especially granulocytopenia. This apparently happens very infrequently with propylthiouracil and can be averted. The patient is examined weekly and a weekly or bi-weekly blood count taken. If the WBC falls below 4300 or if less than 45% granulocytes are present therapy should be discontinued. Other rare reactions are drug fever and rash.
  - (b) The second objection is of a technical nature, since the gland may remain hyperplastic and vascular surgical removal is more difficult. Because of this combined therapy using propylthiouracil and iodine is probably the method of choice in preparing patients for thyroidectomy (see below).
- (2) Dosage. Therapy is usually continued and a very definite until B.M.R. is normal. There is no need to rush surgery; there is no danger of escape as with iodine.
  - (a) In severe cases 100-200 mg ( $\frac{1}{2}$ -3 gr) q.i.d. is generally adequate.
  - (b) In severe cases especially with very large glands larger doses may be necessary.
  - (c) In milder cases 100 mg ( $\frac{1}{2}$  gr) b.i.d. may be used. Although the larger doses are not more harmful.
- (b) Methimazole (N.N.R.) is almost the same as propylthiouracil in mode of action and dosage. Toxic reaction may be more frequent.
- (c) Mithimazole (N.N.R.) (Tapazole®). The action of this drug is similar to that of the thiouracils. The dosage is about  $\frac{1}{10}$ - $\frac{1}{15}$  that of propylthiouracil. The average dose is 10-15 mg ( $\frac{1}{8}$ - $\frac{1}{4}$  gr) v.o. 3-4 times. The smaller dosage is no guarantee against toxic reactions which may be more common with this drug than the thiouracils.
- (d) Iodothiouracil (Itrumil®). This iodinated thiouracil is claimed to be non-goutogenic. Although some favorable reports have been published the others have been reports of gradual escape while on the drug as well as cases of postoperative crisis. Dosage 100-200 mg ( $\frac{1}{2}$ -4 gr) t.i.d. to q.i.d.

## 372 Hyperthyroidism

- 3 Combined propylthiouracil iodine preparation The advantage of this method is that one obtains the complete inhibition of thyroid secretion with the involuting effect of iodine. This can be given in 2 ways

- Propylthiouracil followed by iodine. This appears at present to be the method of choice. Begin therapy with propylthiouracil about 10-21 days before surgery is contemplated (usually B.M.R. about +20) begin the iodine and continue for 1 week after surgery.
- Concomitant administration of the 2 drugs from the start in dosages as for the individual drugs i.e. 100-200 mg ( $1\frac{1}{2}$ -3 gr) propylthiouracil q.i.d. and Strong Iodine Solution U.S.P. Aqueous Solution of Iodine B.P. (Lugol's solution) 10-15 drops daily.

### B Continuous Propylthiouracil Therapy (Medical Treatment)

Control of hyperthyroidism with propylthiouracil alone without surgery has been advocated by some

- The advantage is that it avoids the risks and postoperative complications of surgery e.g. myxedema hypoparathyroidism.
- The disadvantage is the possibility (highly improbable) of toxic reactions (see p. 371) plus the necessity of watching the patient carefully for signs of hypothyroidism. Since the advent of propylthiouracil it appears that the possibility of toxic reactions is slight. The patient must report to the physician if fever, sore throat or dermatitis develop.

#### 3 Dosage

- Begin with 100-200 mg ( $1\frac{1}{2}$ -3 gr) t.i.d. to q.i.d. and continue this dosage until B.M.R. is normal and all signs and symptoms of the disease have subsided then place the patient on a maintenance dose of 50-75 mg ( $\frac{3}{4}$ -1 $\frac{1}{4}$  gr) daily keeping check on the B.M.R. or protein-bound iodine to avoid hypothyroidism.
- An alternative method is to continue with doses of 100-200 mg ( $\frac{3}{4}$ -3 gr) t.i.d. to q.i.d. This will bring the patient to hypothyroid levels keep his B.M.R. or protein-bound iodine normal with thyroid. (This may be the preferred treatment of exophthalmic goiter see p. 373)
- Duration of therapy. The duration of therapy and recurrence rate have not been completely worked out. However at present it would seem that of the patients kept on propylthiouracil between 6 and 18 months (the dosage slowly decreased) about 50 to 70% will show no recurrence of hyperthyroidism. Increasing the duration of therapy to about 2 years or more does not increase the cure rate.

- ### C Radioactive Iodine ( $I^{131}$ )
- The administration of radiiodine has proved to be an excellent method for ablation of over functioning thyroid tissue. The rationale of treatment is that the radiiodine being concentrated in the thyrocytes will destroy the cells that concentrate it. Its use may be lifesaving in cases of thyroid carcinoma when the cancer tissue can take up iodine. Because special techniques are necessary to measure and handle the  $I^{131}$  the method is still generally limited to larger medical centers. The only objections to date to  $I^{131}$  therapy are the

possibility of carcinogenesis (which has not yet been observed) and the possibility that an early carcinoma which might be removed surgically may remain undetected. Because of the above factors its use should generally be limited to older age groups (50 or above).

**D Cytotoxic Iodine Therapy** In the past this method was used in some mild cases of hyperthyroidism with fair results however because of the danger of escape and because of the discovery of propylthiouracil iodine should be used only for preoperative preparation.

**E X-ray Therapy** Has been used in skilled hands with good results as a substitute for surgery but because of the time necessary to obtain complete effect (3-6 months) this method of therapy should be reserved for selected cases in which rarely iodine is available.

#### **F General Management**

1. **Rest** The patient with hyperthyroidism should be treated especially in severe cases and in preparation for surgery. With the advent of propylthiouracil mild cases as being treated as ambulatory patients. However severely bedridden patients require recovery.

2. **Diet** Diet should be high in calories proteins and vitamins. The patient who consumes great quantities of food and gets generally in negative nitrogen balance and needs extra foods and vitamins because of their increased metabolic needs. Supplemental vitamin B complex should generally be employed.

3. **Sedation** When first seen the patients are often very nervous. Sedation is always helpful and very largely qualitative may be necessary in control of symptoms. Phenobarbital U.S.P. Phenobarbital B.P. 100 mg (1/2 gr) 3-4 times daily may be necessary.

4. **Treatment on propionates** This drug has been shown to be of value in restoring positive nitrogen balance in these patients. Give 25-50 mg (3/8-3/4 gr) 1-3 times daily or 2-3 times per week. Do not use methylglucosterate in hyperthyroidism as this aggravates the condition and seems to aggravate the hyperthyroidism.

#### **G Treatment of Complications**

1. **Exophthalmos** The nature of exophthalmos is still unknown. Although it may be due to excessive secretion of thyrotrophic or other anterior pituitary hormone the evidence is still inconclusive. It has been shown that exophthalmos is due to edema and later vitreous infiltrations of the periorbital tissues (muscle, connective tissue, etc.). Removal of thyroid secretion (by extirpation or administration of propylthiouracil) does not necessarily help the condition and may aggravate the existing malignant exophthalmos. It has been suggested that this is due to the fact that the thyroid secretion acts as an inhibitory factor on the anterior pituitary removal of the gland allowing the anterior pituitary to secrete more hormone and aggravate the condition. Some have suggested that exophthalmos occurs with hyperthyroidism because the thyroid secretion in hyperthyroidism may be abnormal and be a factor.

abnormal secretion does not have any pituitary depressing effect. Therefore it would seem rational to treat this condition by giving thyroid orally.

- a **Thyroid dosage** - Immediately after surgery or after B M R has returned to almost normal ( $\pm 20\%$ ) with propylthiouracil therapy begin giving thyroid 100-200 mg ( $1\frac{1}{2}$ -3 gr) daily. Give dosage adequate to maintain B M R at about  $\pm 20\%$ . This therapy should be used whenever there is a tendency for progression of the exophthalmos although it is not always effective.
- b **Physical protection of eyes** - Dark glasses, protection from dust, eye shields, tarsorrhaphy and other measures may be necessary. Ophthalmological consultation should be requested.
- c **ACTH or adrenal steroids** - The use of these agents in large doses has been proposed. In some cases they have proved helpful. They probably act by reducing the inflammatory reaction which occurs in the peri-orbital tissues.
- d **Surgery of malignant exophthalmos** - Every patient with exophthalmos should have actual and periodic measurements made with an exophthalmometer. One should not rely upon clinical judgment to determine whether or not exophthalmos is present or progressing. In severe progressive cases where corneal edema, limitation of extraocular muscle movements and falling vision occur it becomes practically a surgical emergency to save the eyesight. The operation of choice is orbital decompression.

## 2 Cardiac complications

Whether or not thyrotoxicosis itself can cause heart disease is still unsettled; however a number of cardiac complications are at times associated with hyperthyroidism.

- a **Tachycardia** - Some degree of tachycardia is always found if normal rhythm is present in thyrotoxicosis. This requires only the treatment of the thyrotoxicosis.
- b **Congestive failure** - This tends to occur in long standing thyrotoxicosis especially in the older age groups. Therapy is the same as for congestive failure from any cause. Digitalis seems to be effective in congestive failure associated with thyrotoxicosis (see p 187).
- c **Auricular fibrillation** - May occur in association with thyrotoxicosis. Treat as any other auricular fibrillation but do not try to convert the auricular fibrillation in a toxic patient. Most cases will revert to normal rhythm soon after toxicity is removed. However if fibrillation remains for 2 weeks after surgery or for 2-4 weeks after B M R has returned to normal using propylthiouracil therapy one should consider use of quinidine to convert to a normal rhythm (if no contraindications are present) (see p 200).

**Crisis or storm** - Fortunately this condition is rare with modern forms of therapy. It occurs now mainly with inadequately thiouracil iodine treated patients immediately after subtotal thyroidectomy. It is characterized by

hyperpyrexia tachycardia and C N S hyperirritability and delirium. The cause is uncertain but absolute or relative adrenal cortical insufficiency may be important.

- a General treatment: Attempt to control the hyperpyrexia with cold packs and the hyperirritability with sedation.
- b Specific measures: There is no certain specific therapy. However the use of large doses of whole adrenal cortical extract, cortisone and corticotropin (ACTH) may be life saving. The administration of large doses of sodium iodide 1-2 Gm (15-30 gr) i.v. and repeated every 12-24 hours has been advocated.

## DISORDERS OF CALCIUM AND PHOSPHORUS METABOLISM

### NORMAL CALCIUM AND PHOSPHORUS METABOLISM

#### Calcium

- A Intake: Calcium is ordinarily derived from the diet. Average adult dietary intake is 0.5-0.8 Gm ( $7\frac{1}{2}$ -12 gr) daily and is normally adequate. During pregnancy and lactation the requirements are higher, the range being 1.5-3.0 Gm (23 $\frac{1}{2}$ -45 gr) daily.
- B Absorption: Calcium is absorbed in the small intestine.
  - 1 Factors influencing calcium absorption:
    - 1 Vitamin D: Needed for proper absorption.
    - 2 Presence of fatty acids or certain minerals (magnesium, potassium) may interfere with calcium absorption.
    - 3 pH of intestine: Increased acid favors absorption.
    - 4 Diseases of the GI tract (e.g. chronic diarrhea, pancreatic deficiency) which disturb motility and interfere with absorption.

#### Calcium metabolism

- 1 Blood: Calcium exists in plasma in 2 fractions: a diffusible fraction (45-50%) containing the ionizable active material and a non-diffusible which is bound to the globulins. When blood calcium values are reported, these are the total (diffusible and non-diffusible) calcium and may be low when protein level is low, but the physiologically active ionizable portion may be normal. Therefore, always determine serum protein when serum calcium is determined.
- 2 Bone: Bone is a very actively metabolizing tissue. There is constant breakdown or resorption (osteoclastic activity) and constant new bone matrix formation (osteoblastic activity) and constant calcification of this matrix. The activity of building up the new bone (osteoblastic activity) is associated with the presence of alkaline phosphatase enzyme and its liberation into the blood. This enzyme is increased when osteoblastic activity is increased.
- 3 Excretion: Calcium is excreted in the urine and stools. Most of the blood calcium is derived from unabsorbed dietary calcium and varies with calcium intake and absorption. The urinary calcium varies with the amount absorbed, but the excretion is not as great as with the stool calcium.

**HYPOPARATHYROIDISM (Unknown Cause code No 820 x10)  
(Injury due to Operation code No 820-415 x)**

A deficiency of parathyroid hormone usually occurring post operatively following thyroidectomy or surgery for parathyroid tumor or hyperplasia. It is characterized by muscle weakness, irritability, tetany, a low blood calcium, high or normal blood phosphorus, normal phosphatase and normal bones by x ray (except after removal of parathyroid tumor). Cataracts may occur particularly in young persons.

Treatment

A Emergency Treatment for Acute Attack (Hypoparathyroid Tetany) Usually postsurgical and requires immediate treatment

1. Calcium Chloride U S P B P 5-10 cc (1 2½ dr) of 10% solution I V slowly until tetany ceases or Calcium Gluconate Injection U S P B P III 30 cc (1½ oz) of 10% solution I V may be given. 10-50 cc of either solution may be added to 1000 cc of 5% glucose in water or saline and administered by slow I V drip. The rate should be so adjusted that hourly determination of urinary calcium by means of the Sulkowitch test will be positive.
2. Parathyroid Injection U S P 50-100 units (½-1 cc) I M or subcut 3-5 times daily as necessary to prevent tetany. Do not use parathyroid hormone for over one week because refractory state tends to develop rapidly. Use only as long as absolutely necessary. Actually parathormone is rarely ever used. It is not very practical and usually not necessary.
3. Calcium salts should be given orally as soon as possible: calcium gluconate 4 Gm (60 gr) q i d; calcium lactate or calcium chloride 2-3 Gm (30-45 gr) q i d.
4. Dihydrotachysterol (A T 10) should be given as soon as oral calcium is begun. Begin with oral dose of 4-10 cc (1 2½ dr) of oily sol (1-25 mg per cc) daily for first 2-4 days then reduce dose to 1-2 cc daily for 1-3 weeks and then determine patient's maintenance requirements.

B Maintenance Treatment

1. High calcium low phosphorus diet (omit milk).
2. Calcium salts as above may be continued (except calcium chloride).
3. Dihydrotachysterol (A T 10) 1½-1 cc daily or 3 times weekly to maintain blood calcium at normal level.
4. Calciferol U S P 2-5 mg daily. In some cases up to 7 or 8 mg calciferol daily may be substituted for dihydrotachysterol. Vitamin D action is probably similar to that of dihydrotachysterol and it can certainly be substituted adequately clinically. The initial action of vitamin D appears to be slower. However, the cost to the patient is less than using dihydrotachysterol and the margin of safety is probably greater. Regulate the dose by daily Sulkowitch test which should run 1-2+.
5. In some patients probenecid (Benemid®) in doses of 2-4 Gm (30-60 gr) daily has been shown to block the tubular reabsorption of phosphate and hence to lower serum phosphorus.

and help restore serum calcium levels to normal in this condition

- 6 Aluminum hydroxide gel may be employed to help lower the serum phosphate (see p 302)

### OSTEOPOROSIS (Senile Osteoporosis code No 200 796)

Osteoporosis occurs most commonly in postmenopausal patients. It is also associated with other conditions leading to general tissue atrophy e.g. malnutrition particularly due to low protein intake Cushing syndrome excessive use of corticotropin (ACTH) or cortisone distal atrophy (where stimulus to osteogenesis is absent) craniogly or may be idiopathic

The first complaint is usually backache. There is a history of occurrence of rarefaction especially in lumbar vertebrae and distal pelvis and often collapse and fracture of vertebral bodies. Other pathological fractures may occur. The blood calcium phosphate and alkaline phosphatase are normal

#### Treatment

A Specific measures vary with the cause but combined hormone therapy is usually indicated

- 1 Postmenopausal (mostly in females)

- a Estrogens may be effective in stimulating osteoblasts. Before beginning estrogen therapy in a postmenopausal woman perform a careful pelvic examination to rule out neoplasia or other abnormality and warn patient of a relative that vaginal bleeding may occur. Administer oestradiol 1 mg daily 10 or 15 or 20 mg of each month and then repeat cycle

(1) Diethylstilbestrol USP Stilbestrol BP 0.5 2.5 mg daily as tolerated

(2) Ethinyl Estradiol N N R 0.02 0.05 mg daily as tolerated

- b Testosterone For its protein anabolic effect and hence its tendency to lay down bone matrix testosterone may be added in addition to estrogens. Give 10-20 mg methyl testosterone orally. Avoid overdosage in females excessive use may cause appearance of male secondary sexual characteristics. However these will regress if therapy is stopped

- 2 Old age and idiopathic. A postmenopausal woman's dose is one and a half times should be used in both male and female

- 3 Patients with malnutrition. Adequate diet is of great importance. However the hormones may be used as above if response is distal osteoporosis

- 4 Cushing's disease (see p 383)

#### \* General

- 1 Should be high protein high-calcium (milk and milk products etc). Vitamin supplements especially vitamin D 4000-5000 units daily may be given also

- 2 Activity. Patients should be kept active. If bed rest is unavoidable an analgesic should be used



## OSTEOMALACIA (code No 200 7642)

Osteomalacia results from calcium deficiency due to any cause in ad its these include vitamin D lack or resistance (rare) and sprue syndrome or pancreatic disease. Chronic renal disease may also produce osteomalacia but more generally produces secondary hyperparathyroidism.

Rickets (code No 010 764) is the childhood type due to inadequate intake of vitamin D. Laboratory findings include a low or normal serum calcium or a low or normal serum phosphorus (except in renal disease where it may be elevated) and most characteristically an elevated phosphatase.

### Treatment

#### A Specific Therapy

- 1 Rickets Vitamin D even in small doses is specific. 2000-5000 units daily are adequate.
- 2 Adult osteomalacia and Milkman's syndrome Vitamin D is specific but very large doses are necessary to overcome the absorption defect. Give until an effect is noted on blood calcium. Usual dose is 25 000-100 000 units daily. Doses up to 300 000 units daily may be necessary but if the doses are over 100 000 daily they must be used cautiously.
- 3 Pancreatic insufficiency (see p 289) Adequate replacement therapy is of paramount importance. High calcium intake and vitamin D 2000-100 000 units daily are also of value.
- 4 Sprue syndrome Folic acid and vitamin B<sub>12</sub> appear to be of value (see p 226).
- 5 Some rare forms of renal disease Treatment is aimed at the altered renal physiology.

B General Measures High calcium diet and calcium gluconate, calcium lactate or calcium chloride 5-20 Gm (1-5 dr) daily.

## ADRENAL CORTEX

### ADDISON'S DISEASE (Adrenocortical Insufficiency)

(Due to Tuberculosis code No 860 123 x)

(Undetermined Cause code No 861 782)

A disease due to lack of secretion of adrenal cortex caused by tuberculous destruction of the gland, surgery or undetermined factors. It is manifested by asthenia, anorexia and other GI disturbances, hypotension and pigmentation, usually brownish of the skin and mucous membranes. This pigmentation is mainly an accentuation of already pigmented areas and a deposition of pigment in skin creases.

The laboratory findings include a low blood sugar, increased insulin sensitivity, low blood sodium and chloride, elevated potassium, elevated N.P.T. and a positive water test. There is a decrease of 17-ketosteroid excretion in the female and lack of response to adrenocorticotrophic hormone in primary Addison's disease.

## Treatment

## A Specific Therapy (Chronic Case)

- 1 Corticosteroids The drugs of choice at present for Addisonian patients are well maintained on dosages of 25-35 mg (1/2-3/4 gr) daily given in divided doses 3 to 4 times daily orally. On this dose most of the metabolic abnormalities are corrected. Some patients however do not obtain sufficient salt retaining effect and they require some D O C A supplement or extra dietary salt.
- 2 Desoxycorticosterone Acetate USP Desoxycortone Acetate (D O C A) The effect of this drug is limited to controlling electrolyte balance and has no other significant metabolic effect.
- a IM administration of D O C A may be used initially but is rarely necessary. The usual dose for a supplement is 1-4 mg daily. When the response has been adequate (see below) change to buccal use.
- b Buccal use of D O C A (hard compressed tablets containing 2 mg of D O C A in carbowax). One tablet daily at most 1 tablet twice daily will give adequate supply in adults. The tablet is placed between cheek and teeth and allowed to dissolve. The dosage of subcutaneous Desoxycortone trimethylacetate 25-75 mg IM once monthly may be used instead of D O C A (25 mg IM once monthly is roughly equivalent to 1 mg D O C A in oil per day).
- CAUTION** Whenever using D O C A avoid overdosage. Also do not have patient on low potassium diet when giving D O C A. If patient may develop signs of potassium deficiency.

- 3 Sodium chloride in larger dose (3-20 Gm daily) may be used to supplement sodium in the absence of D O C A.

## B General Measures

- a High carbohydrate and high protein intakes are very important.
- b Frequent small feedings rather than 3 large ones tend to be better tolerated.
- 2 Avoid exposure to infection and vigorous activity.
- 3 Treat one Methyltestosterone 10-20 mg daily orally or 1 testone propionate in oil 10-25 mg IM three times weekly if not helpful for its protein anabolic effect as well as the anabolic effect of well being if indicated in the debilitated patient.

## Criteria of Adequate Therapy and Overdosage

- 1 Restoration of blood pressure to normal. May require up to 3-4 months with adequate therapy.
- 2 Maintenance of normal fasting blood sugar level.
- 3 Restoration of plasma electrolytes to normal levels.
- 4 Weight gain (usually due to fluid).
- 5 Improvement of appetite and of general well-being.

6 Increase in size of heart to normal

- B Overdosage** Must be watched for and avoided very carefully especially in patients with cardiac or renal complications
- 1 Signs and symptoms of cortisone overdosage (see p 423)
  - 2 Development of dependent edema or excessive weight gain
  - 3 Development of hypertension
  - 4 Increase of diameter of heart above normal
  - 5 Development of signs of potassium deficiency (weakness followed by loss of muscle power and finally paralysis) especially if patient is on a low potassium diet

### Treatment of Adrenal Crisis (Acute Adrenal Insufficiency)

The adrenal crisis is an emergency. The patient must be treated vigorously and observed constantly until well out of danger. *Overtreat rather than undertreat.*

#### A Severe Crisis

##### 1 Emergency treatment

- a Anti shock measures. Combat shock by use of appropriate adjunctive measures (see p 31) especially plasma, vasopressor drugs and oxygen. Do not use narcotics or sedatives.
- b Hydrocortisone. Administer hydrocortisone free alcohol (Infusion Concentrate Hydrocortone®) 100 mg in 1000 cc 5% glucose in physiological saline solution by I V infusion over a period of 1 to 3 hours. An additional 50-100 mg of hydrocortisone may be added to subsequent infusions during the first 24 hours if necessary. (If parenteral hydrocortisone is not available give aqueous adrenal cortical extract 25-50 cc I V immediately and follow with 100-200 cc aqueous adrenal cortical extract in 1000 cc saline dextrose infusion.)
- c Cortisone acetate. Give initial cortisone acetate 10-25 mg I M in four different sites (total 40-100 mg). Follow with single injections of cortisone 25-50 mg I M every 6 hours and gradually lengthen intervals of administration to 25 mg every 8 hours.
- d Subsequent parenteral fluids. After the first I V infusion mentioned above is completed it should be followed immediately by 1000 cc 10% glucose in physiological saline solution (including additional hydrocortisone as mentioned above). The total fluids in the first 24 hours and daily thereafter should not be greater than 3 liters in order to avoid excessive administration of sodium.
- e Anti infective measures. If infection is present treat intensively with indicated antibiotic.

- 2 Convalescent treatment. When patient is able to take food by mouth place on oral cortisone 12.5-25 mg every 6 hours and reduce dosage to maintenance levels as needed.

- II Moderate Crisis** If the patient's physical condition does not appear to be critical and is not associated with a significant degree of shock the treatment outlined above may be modified by appropriate reduction in dosage. However it is generally best to overtreat the patient in moderate crisis during the first 24 hours rather than risk undertreatment.

#### C Complications Arising During Course of Treatment of Crisis

- 1 Overhydration. Overhydration usually due to sodium retention may result in cerebral edema (with unconsciousness or convulsions) or pulmonary edema. Withhold sodium and fluids temporarily and treat for this condition (see p 21).
- 2 Hypokalemia. Flaccid paralysis with low serum K usually occurring on 2-4th days of treatment may be treated with K salts (see p 21).
- 3 Hyperpyrexia. This complication is rare with present intravenous method (See p 30 for therapy).
- 4 For the complications of adrenal steroid therapy (e.g. psychotic reactions) see p 423.

**CUSHING'S SYNDROME (Adrenocortical Hyperfunction)**  
 (Pituitary Basophilism code No 841 7753)  
 (Adrenocortical Hyperfunction code No 861 7813)

A chronic and if untreated usually fatal disease due to hyperfunction of the adrenal cortex caused by primary hyperplasia or adenoma or carcinoma of the adrenal cortex. The underlying physiological disturbance is probably an overproduction of the labile (glucose stimulating) corticosteroids. A retention of the adrenal cortex. It is manifested by a thinning of the skin and trunk osteoporosis, hypertension, abdominal striae, weight gain, susceptibility to infection, and inulin resistant diabetes. The adrenal dysplasia (in the case of hyperplasia) and in the case of adenoma or carcinoma, a tumor of the adrenal cortex. The tumor may be removed surgically. A low eosinophil count is found in the blood usually below 50 per cu mm.

Treatment

In evaluating the results of therapy, it is important to note that the disease may be self-limited and spontaneous remission may be expected for the disease may be self-limited.

A Specific Therapy

- 1 Removal of the tumor or total or bilateral resection of both adrenals in the case of diffuse bilateral hyperplasia is the present treatment of choice. Adequate preoperative medication and care of almost importance. The patient should receive all general measures indicated below plus adequate hormonal supplementation where surgery is indicated.
- 2 Preparation with corticosteroids (ACTH) should be carried out well a corticosteroid therapy for bilateral adrenalectomy (see below). The ACTH time is the non-tumorous adrenal cortex which is generally atrophied. Treatment with the usual dose of ACTH should be continued postoperatively for 7-14 days.
- 3 If bilateral adrenalectomy is contemplated, give high doses of corticosteroids 100-300 mg (11-42 g) orally as well as 100-300 mg IM for 3-5 days preoperatively and continue the IM and possibly the oral dosage during and

after surgery After surgery gradually decrease the dose and maintain the patient as a chronic Addisonian patient (see p 381) Because of danger of precipitating heart failure care must be exercised to avoid excessive fluids and sodium

- 2 X ray therapy to the pituitary may be of value only in rare cases of hyperplasia

#### B General Measures

- 1 Diet High protein diet should be given although dietary attempts to correct the negative nitrogen balance are doomed to failure
- 2 Hormones
  - a Testosterone has been of value in reversing the negative nitrogen balance and possibly in suppressing the pituitary secretion of adrenocorticotrophic hormone thereby aiding certain features of the disease (namely asthenia fat distribution osteoporosis and striae) and possibly in prolonging life For this testosterone propionate in oil IM 50 mg daily I M has been found necessary
  - b Insulin is usually of little or no value in controlling the glycosuria and hyperglycemia Insulin is usually unnecessary as the diabetes is quite mild

### VIRILIZING DISEASES OF FEMALES

(Due to Tumor code No 8041)

In the adult the virilizing or masculinizing disease is usually caused by a tumor arising in the adrenal ovary or from cell rests of one of the above tissues It is characterized by excessive hirsutism (especially of male type) amenorrhea enlargement of clitoris deepening of voice excessive musculature and excessive 17 ketosteroid excretion Surgical removal of the tumor is the treatment of choice

Another form of the disease begins in childhood It is due to over production of androgen type hormones from bilaterally hyperfunctioning adrenal cortices In many of these patients there may be associated manifestations of hypoadrenocorticism (i.e. excessive salt and water loss and failure to maintain a fasting blood sugar) Treatment with cortisone or hydrocortisone has proved valuable in reducing the activity of the glands (apparently through suppression of endogenous ACTH) and in supplying exogenously needed cortisone Dosages necessary have ranged from 20-50 mg ( $\frac{1}{4}$ - $\frac{3}{4}$  gr) daily orally in divided doses Some investigators feel that the same dose of cortisone acetate by the I M route may be more efficacious in this syndrome

Most cases of excessive hirsutism in females are not due to endocrine disease but to hereditary or racial factors and should not and cannot be treated by any internal medications or surgery

### HYPOGONADISM

Hypogonadism is due to a failure of the sex glands to elaborate sufficient quantities of their hormones to bring about or maintain the of secondary sexual characteristics

## MALE HYPOGONADISM

### Etiology

Failure of the gonads may result from a variety of causes. This failure may be primary (i.e., due to testicular disease) or secondary to malfunction of other glands, most often the pituitary or thyroid.

- A Testicular Hypogonadism (Eunuchoidism) (code No. 755.787)
- B Male Climacteric (code No. 808)
- C Congenital Hypoplasia of Testicles (code No. 755.016)
- D Degeneration of the Testes Due to Infection (code No. 755.100 B)
- E Necrosis of Testis due to Trauma (code No. 755.400.1)

### Diagnosis

The physiological diagnosis of the etiology of hypogonadism (e.g., primary or secondary) is usually based on laboratory tests.

Type of Hypogonadism	Urinary LH Ketosteroids	Urinary Gonadotropins
Primary	Low or normal	Elevated
Secondary Pituitary	Usually low but may be normal	Very low
Adrenal Insufficiency	Low or normal	Low or normal. Not generally a low pituitary type
Thyroid (Hypothyroid?)	Low or normal	Low

Many clinical syndromes have been described but basically they all fall into one of two categories. The differences are outlined below.

- A Prepubertal Type Should not be diagnosed before 18 years of age. This is a failure of development of normal testicular function and is manifested by small or absent testicles, small penis, lack of development of axillary and pubic hair, lack of masculine development, often younger bone age than chronological age.
- B Postpubertal Type Loss or cessation of normal testicular function (usually traumatic infection, x-ray and male climacteric). Apparently the cessation of testicular function in the male is not generally as acute and tends to occur much later in life than the cessation of ovarian function in the female. However, at least in some men there is a rather rapid cessation with the development of symptoms as in male menopause (see p. 388).

### Treatment

Testosterone (the male sex hormone) is the drug used for replacement therapy in this male. (For preparations available see p. 423.)

- A Prepubertal Hypogonadism Adequate testosterone therapy can

make these individuals into normal adult males in every way except that they cannot produce sperm. These patients must be placed on testosterone and maintained for life on adequate doses of testosterone. There is little evidence that any pituitary substance or gonadotropin is of significant value in treating these patients (see pp 412-413).

- 1 Any of the testosterone preparations may be used but the free testosterone pellet implantation in experienced hands seems to be most useful. The dosage (number of pellets) is as follows: 300 mg (5 gr) is the minimal but an effective dose. Average is about 600 mg (10 gr). For maximum effect about 900 mg (15 gr) are implanted. The pellets are implanted subcutaneously with a trocar or into a pocket made by blunt dissection. Either the inferior scapular area or anterior thigh area is used. Testosterone pellets remain and are effective for 3-4 months and then must be replaced.
- 2 An alternative method is the oral or sublingual administration of methyltestosterone. This requires daily administration of the drug to the patient and entails all the difficulties of prolonged oral administration. Dosage varies with various individuals but 10-25 mg ( $\frac{1}{16}$  -  $\frac{3}{8}$  gr) daily orally is usually adequate dosage to cause maturation and virilization and maintenance of this state. Evidence now indicates that there is no advantage of buccal over oral administration.
- 3 Long acting testosterone preparations. Testosterone cyclopentylpropionate (Depo testosterone®) 250-500 mg IM every 2-4 weeks may also be employed.

- II Postpubertal. Oral use of methyltestosterone is probably the method of choice. The dosage necessary to control symptoms and to aid in overcoming the protein loss and debility of age is often as low as 10-20 mg ( $\frac{1}{16}$  -  $\frac{1}{8}$  gr) daily. This dose may be used for a short period of time to control symptoms or may be continued indefinitely for control of symptoms and for its protein anabolic effect.

## FEMALE HYPOGONADISM

The most common symptom of female hypogonadism is amenorrhea. However, most cases of relative amenorrhea are not due to hypogonadism.

### AMENORRHEA (code No 761)

#### Etiology

No maturation depends on many factors. From the functional point of view there must be an intact pituitary-gonad-uterine axis. Any break in the cycle of production of the various pituitary or ovarian hormones concerned with normal menstruation or the lack of an endometrium capable of response to ovarian hormones will result in amenorrhea. Treatment is based in general measure on the level at which the disturbed physiology exists.

# Dagnosis

The usual order of special diagnostic steps and their interpretation follows

1. Test for pregnancy: Administer 10-25 mg (95-300 µg) of progesterone orally daily for 5 days. If bleeding occurs within 48-96 hours after the last dose, it is indicative of endogenous progesterone production. If no bleeding occurs, this indicates a lack of endogenous progesterone production.
2. Test for thyroid function: Administer 1 mg of thyroxine daily for 10 days. If bleeding occurs within 72-96 hours after the last dose, it is indicative of thyroid dysfunction. If no bleeding occurs, it is indicative of normal thyroid function.

3. Test for pituitary function: Administer 1 mg of thyroxine daily for 10 days. If bleeding occurs within 72-96 hours after the last dose, it is indicative of pituitary dysfunction. If no bleeding occurs, it is indicative of normal pituitary function.
4. Test for gonadal function: Administer 1 mg of thyroxine daily for 10 days. If bleeding occurs within 72-96 hours after the last dose, it is indicative of gonadal dysfunction. If no bleeding occurs, it is indicative of normal gonadal function.

5. Test for adrenal function: Administer 1 mg of thyroxine daily for 10 days. If bleeding occurs within 72-96 hours after the last dose, it is indicative of adrenal dysfunction. If no bleeding occurs, it is indicative of normal adrenal function.
6. Test for liver function: Administer 1 mg of thyroxine daily for 10 days. If bleeding occurs within 72-96 hours after the last dose, it is indicative of liver dysfunction. If no bleeding occurs, it is indicative of normal liver function.

7. Exploratory laparotomy: Will help with diagnosis if all the above tests are negative.

## Treatment

The aim of therapy of amenorrhoea is not to cause the appearance of menstruation (except psychological reasons) but to stimulate reproductive function.

1. Primary amenorrhoea: (No previous menstruation) Treatment depends on underlying cause. If due to constitutional delay, treatment is unnecessary. If due to organic pathology, treatment is required.
2. Secondary amenorrhoea: (Previous menstruation) Treatment depends on underlying cause. If due to organic pathology, treatment is required. If due to functional causes, treatment is unnecessary.

3. Specific treatment: It is not necessary in all cases of amenorrhoea. It is especially indicated in cases of organic pathology. Treatment should be given in the form of hormone therapy. The aim is to stimulate the hypothalamic-pituitary-gonadal axis. Treatment should be given in the form of hormone therapy. The aim is to stimulate the hypothalamic-pituitary-gonadal axis.



not (see p 413) Employ estrogen alone or in combination with progesterone (see p 422)

(2) With high urinary gonadotropins Gonadotropins are likewise of no value treat as above

## 2 General measures

- a Adequate diet and dietary treatment as needed to correct abnormal deviation of weight
- b Psychotherapy in cases of emotional disturbances
- c Correction of anemia (see p 219)
- d Correction of any other metabolic abnormality (e g hypothyroidism)

## MENOPAUSE

### Etiology

Failure of the ovaries may result from several causes most common of which are the natural menopause and menopause due to surgical removal or to x ray The failure may be secondary to mal function of other glands most often the pituitary or thyroid

A Menopausal Syndrome (code No 805)

B Artificial Menopause Due to Roentgen Rays (code No 788 471)

■ Hypofunction of Ovary Due to Unknown Cause (code No 788 x10)

### Diagnosis

The menopause is due to a loss of ovarian function and is manifested by vasomotor disturbances (e g hot flashes) nervousness emotional instability and amenorrhea Abnormal uterine bleeding may occur in the natural menopause before the ovaries atrophy

### Treatment

A Natural Menopause The menopause in the female is characterized by at least 2 important factors physiological failure of ovarian function which occurs rather rapidly in the female and psychological recognition of the fact that reproductive life is at an end Many believe that as a result of this there is a marked change of life an implication that in addition to cessation of reproductive function there is complete alteration in one's way of life sexual activities personal interrelationships etc This latter belief is entirely erroneous Most women go through the menopause without any difficulty in fact without symptoms However in those having symptoms one must carefully evaluate the role of the physiological and psychological factors before beginning any therapy Most cases will have a mixture of physiological and psychological factors

- 1 Physiological aspects (estrogen therapy) There are certain symptoms that seem undoubtedly to be due to the cessation of ovarian function the most prominent being vasomotor instability (e g flushing) Another may be the feeling of tension especially fullness in the head In women who suffer primarily from symptoms of cessation of ovarian function use of estrogens is indicated The dosage and method of administration used depends on whether or not the patient is still menstruating

- a If patient has regular periods she probably is manufacturing sufficient estrone and does not need therapy
- b If cycles are very irregular and the patient suffers from menopausal symptoms, estrogens given in cyclical fashion may be helpful. Begin estrogens about 5 days after onset of last menstrual period and continue in a cyclic fashion. Ethinyl Estradiol N N 0.05 mg or Diethylstilbestrol U S P Stilboestrol B P 0.5 mg by mouth daily except the first 5 days of each month. This is simple for patient to remember.
- c If patient has become amenorrheic there is no reason to give estrogens in dose large enough to reinstitute menses but only to control symptoms.
- d Duration of therapy. This has not been standardized and must be adjusted to the individual case. Three months to 1 year is usually sufficient.
- e Maintenance dose for life. Because of the anabolic effect of estrogens and because of their known beneficial effect on skeletal metabolism, estrogen therapy has been recommended for the duration of life for women beyond the menopause. The advisability of this practice will undoubtedly not be settled for a long time.

If a patient is on long term estrogen therapy she should keep a permanent record of her dosage schedule and bleeding. Whenever bleeding occurs that is not on schedule (during withdrawal phase) an investigation for tumor should be instituted. Vaginal cytological examination should be carried out routinely every 6 months or 1 year.

- 2 Psychological aspects. Many of the symptoms of the menopause are undoubtedly psychological. The most common symptom is anxiety, more severe emotional disorder may occur.

a Sedation. Phenobarbital U S P Phenobarbitone B P 15 mg (1/4 gr) t i d q i d

b Psychotherapy. Simple explanation and reassurance that their lives need not be changed because of the menopause are adequate in most patients. In the more severe cases however, the aid of a psychiatrist may prove necessary.

- 3 Gynecological and X-ray Mammography. These cases differ from the normal menopausal changes only in the abruptness and severity of the symptoms. Both physiologically and psychologically. In these patients it is advisable to help them patently live as normal a life as possible. If the patients can be made to have normal periods and if they understand that their menstrual cycle will go on unchanged they usually make suitable adjustments. Estrogen therapy is a for natural menopause (see above).

#### Complications of Postmenopausal Hypogonadism and Treatment

A Osteoporosis (see p. 379)

- B Small Tablets. Give Diethylstilbestrol U S P Stilboestrol B P 0.5 mg (1/40 gr) or other estrogens (122) daily orally. Stilbestrol vaginal suppositories containing 1 mg (1/40 gr) may be used daily for 10 to 14 days while continuing oral stilbestrol.

## MENORRHAGIA (code No 785 x20) FUNCTIONAL UTERINE BLEEDING

With the gradual failure of ovarian function excessive bleeding at the time of the menses is a common occurrence. This has been shown to be due to prolonged hypoestrogenic effect without concomitant progesterone production.

Another type of functional uterine bleeding occurs most commonly in young women. Here a hyperestrogenic effect is responsible. In any case of prolonged and unusual bleeding suspect and rule out neoplasms of the uterus.

Treatment consists of administration of progesterone 100 mg (1 $\frac{1}{2}$  gr) orally or 10-15 mg ( $\frac{1}{16}$  -  $\frac{1}{4}$  gr) I.M. daily for 5 days. This converts the proliferative endometrium into a secretory one with complete shedding when the progesterone is withdrawn.

## OBESITY (code No 007)

(Due to Excess Food code No 010 70x)

(Undetermined Cause code No 010 70y)

Obesity may be defined as an increase in weight of over 10% above normal due to generalized deposition of fat in the body.

Normal weight is difficult to determine for average clinical practice; however, normal as defined by the standard age, height and weight tables is satisfactory (see table p. 572).

From a metabolic point of view all obesity has a common cause: intake of more calories than are required for energy metabolism. The reasons for differences in the energy utilization of various individuals whereby that one person can utilize his calories more efficiently than another is unknown. Although most cases of obesity are due to simple overeating, a few endocrine disorders lead to specific types of obesity (e.g., Cushing's syndrome and hypothalamic lesions), but these conditions are rare. Hypothyroidism on the other hand is not necessarily associated with obesity.

### Treatment

Specific weight-reducing chemical agents and hormones singly or in combination are either ineffective or hazardous and have no place in the treatment of exogenous obesity.

A. Diet (see p. 572). Diet is the most important factor in the management of obesity. There are a number of points to consider:

1. Calories. In order to lose weight it is necessary to decrease the intake to below the caloric requirements of the individual (see p. 45). One can determine a very approximate average weight loss per day with a given diet by the following formula:

$$\frac{\text{Approximate Caloric Requirement Per Day} - \text{Number of Calories in Diet}}{4000} = \text{Weight Loss in lbs. / Day}$$

The number of calories per day to prescribe for a patient varies with age, occupation, temperament and the urgency of the need for weight reduction. A daily caloric intake of 800-1200 Calories is satisfactory for a reducing diet.

There is no evidence that supervised rapid weight loss is harmful. It has been shown that with adequate protein intake nitrogen balance can be maintained on 350-450 Calorie per day. In the severely restricted diet ketonuria may appear. It is usually very slight after the first few days, however, and acidosis has never been observed. In addition since the patients realize they are on a diet they often will adhere more willingly when they show rapid weight loss than when the results seem to be slow in appearing.

- 2 Protein. A protein intake of at least 1 Gm/kg (0.45 Gm/lb) should be maintained. If it is necessary to add protein to the low caloric diet protein hydrolyzate or casein that is free of carbohydrate and fat can be used.
- 3 CHO and fat. To keep the caloric and ketosis down fats are decreased as much as possible. After the protein requirement has been met most of the remaining diet is applied from CHO.

- 4 Vitamins and minerals. Most restricted diet is likely to be deficient in vitamins but adequate in minerals. Therefore any good vitamin preparation should be used to supply the average daily maintenance requirements during the time of weight reduction.

- 5 Sodium restriction. It has been shown that a normal person on a salt free diet will lose from 5-6 lb (2.3 Kg) of weight. This reduction is temporary and the weight will return when salt is added to the diet. The same is true of the bee patient and although an apparently dramatic effect can be obtained with salt free diets it is of no permanent value.

#### Medication

- 1 To decrease the appetite. Amphetamine Sulfate 1.5 gr. It has proved of immense value in aiding patients on reducing regimen by decreasing the appetite and giving a sense of well being. In proper doses it is rarely contraindicated except in cardiovascular diseases especially hypertension and in those patients in whom the drug produces a CNS stimulation. Be aware of its CNS stimulating effect. It is best to use a small dose usually given twice a day in the morning and early afternoon or 12 hours before bedtime. (Benzedrine®) 2 1/2 to 3 mg (1/2 to 3/4 gr) b.i.d. or amphetamine (Benzedrine®) 5 to 10 mg (1/2 to 1 gr) b.i.d. or amphetamine (Benzedrine®) 2 1/2 to 3 mg (1/2 to 3/4 gr) b.i.d.
- 2 Drugs to speed up metabolism. There is no effect, only a temporary speed up metabolism.
- 3 Thyroid. Thyroid has little or no effect in the management of obesity. The low BMR found associated with obesity is merely an artifact caused by the fact that the basal metabolism is decreased. The body's fat stores are relatively poor oxygen consumers. Namely fatty acids are relatively poor oxygen consumers. Actually the basal metabolism is more active than we and hence apparently low BMR results. Greatly caloric requirement of an obese individual is greater than they would be if the same individual were of normal

- 2 Liver glycogen stores become depleted in supplying glucose to the body
- 3 Protein stores begin to break down at an excessive rate to supply glucose (gluconeogenesis)
- 4 Fat becomes the main source of energy
- 5 Fat oxidation is very efficient to the point of ketone body formation ( $\beta$  hydroxybutyric acid and acetoacetic acid)
- 6 The maximum rate of ketone body oxidation by the peripheral tissues is slower than their rate of formation
- 7 As a result of increased ketone body formation and their slow utilization ketone bodies accumulate in the blood and spill over into the urine. Since these substances are acid they are excreted in the urine joined to fixed base ( $\text{Na}^+$   $\text{K}^+$   $\text{Ca}^{++}$  etc.). The accumulation of acid ions and loss of fixed base result in the condition known as acidosis.

### Diagnosis

The typical clinical features in untreated diabetes mellitus are polydipsia, polyphagia, polyuria, and weight loss, but these occur only in the more severe forms of the disease. Because of the varied and nonspecific symptomatology of the disease, the actual diagnosis of diabetes rests upon laboratory evidence. It should be emphasized that all laboratory tests for diabetes are nonspecific and abnormalities of one or all may occur in other diseases (e.g., hyperthyroidism, liver disease). However, in the absence of other diseases, glycosuria and hyperglycemia are diagnostic.

- A Glycosuria. The presence of reducing substances identified as glucose in the urine is excellent presumptive evidence.
- B Hyperglycemia. The finding of an elevated fasting blood sugar and/or abnormal blood sugar level 2 hours after a meal containing 100 grams of carbohydrate or a dose of 100 grams of glucose is almost diagnostic in the absence of other diseases. This test should be performed, however, only after the patient has been on a high carbohydrate diet for at least 48 hours. It is well known that the previous diet influences carbohydrate tolerance. A high fat diet will decrease tolerance (i.e., give diabetic type curve even in normals) and a high carbohydrate diet will increase tolerance.
- C Interpretation of Blood Sugar Tests. A normal fasting blood sugar does not rule out diabetes. If the 2 hour postprandial blood sugar level is over 140, one can be reasonably certain that the condition is diabetes. If the blood sugar level is between 90 and 140, it is necessary to perform a glucose tolerance test to establish the diagnosis.

### TREATMENT OF DIABETES MELLITUS

In order to treat patients with diabetes it is necessary that one be thoroughly familiar with the following:

- 1 Insulin (probably of greatest importance)
- 2 Diet
- 3 Influence of exercise
- 4 Prompt treatment of complications, etc.

Insulin

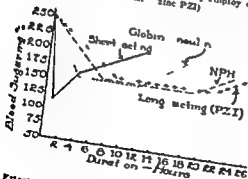
Insulin is utilized clinically to enhance carbohydrate oxidation. This is manifested clinically by noting the lowering of the blood sugar or the lessening or disappearance of glycosuria.

A Duration of Action of Insulin Preparation There are 3 main types of insulin: 1. Short acting insulin Insulin Injection U.S.P. B.P.

2. Regular insulin  
a. Crystalline zinc insulin  
(For clinical purposes the actions of these 2 insulins are identical. Crystalline zinc insulin is preferred only because of its greater purity). These are used mainly in controlling postprandial blood sugar elevations.

3. Long acting insulin Protamine Zinc Insulin Injection U.S.P. B.P. This is useful for lowering the milder hyperglycemia which is present during the remainder of the time between meals.

4. Intermediate acting insulin  
a. Isophane insulin NPH (NPH) A stable mixture with properties much like a 2:1 mixture and has been added to replace PZI in the management of most diabetic patients. It may also be tailored to fit the patient by addition of appropriate amounts of regular insulin. This insulin is similar in action to 2:1 insulin mixture except that its duration of effect is not so prolonged. It is useful in many diabetic patients but it cannot be mixed with short acting insulin. b. Globin insulin with Zinc U.S.P. This insulin may be prepared by mixing a short acting and a long acting insulin in a suitable ratio. By the use of these mixtures one obtains a balance between the immediate effect and the prolonged effect by modifying the mixture. The mixture can divergeously tailor the insulin requirements to the individual needs of the patient. The mixtures usually employed clinically are 2:1 or 3:1 (crystalline zinc PZI).



Effect and Duration of Action of Various Insulins  
(In a Fasting Diabetic)

- (1) Points to remember in use of insulin mixtures
  - (a) Regular insulin must always be withdrawn into syringe before withdrawing PZI (because of protamine excess in PZI)
  - (b) Same unitage of regular insulin and PZI must be used
  - (c) General effect of I/PZI mixtures
    - 1 1 largely PZI effect (little point to this mixture)
    - 2 1 intermediate daytime nitetime effect
    - 3 1 greater daytime effect
- (2) Application of tailored insulin mixtures
  - (a) If glycosuria occurs in all fractional urines increase total insulin mixtures
  - (b) If glycosuria occurs in fractional urines 1 and 2 only (daytime glycosuria) increase regular insulin in mixture
  - (c) If glycosuria occurs in fractional urines 3 and 4 only (nitetime glycosuria) increase PZI in mixture

II Commercial insulin preparations come in various strengths (units/cc) in 5 and 10 cc ampules identified as follows

Potency Preparation	Color of Rubber Stopper	Color of Label
U20 Unmodified regular	Yellow	Yellow
U20 Crystalline	Yellow	Gray and blue or blue gray and yellow
U20 PZI	Not Available	
U40 Unmodified regular	Red	Red
U40 Crystalline	Red	Red and gray
U40 PZI	Red	White label with red printing
U40 NPH	Red	Blue and white
U40 Globin Zinc	Red	Red and brown
U80 Unmodified regular	Green	Green
U80 Crystalline	Green	Green and gray
U80 PZI	Green	White label with green printing
U80 NPH	Green	Blue and white
U80 Globin Zinc	Green	Green and brown
U100 Unmodified regular	Orange	Orange

#### III Administration of Insulin

- 1 Selection of insulin preparation In view of the large number of insulin preparations available there is often great confusion regarding dosage. Therefore it is necessary to place the patient on one type of insulin and have him become familiar with this type. One uses an insulin of such strength that the volume per injection is kept at 0.25 to 0.5 cc. About 80% of patients are able to use U40 insulins.

- 2 **Syringes** In order to aid patient syringes are available calibrated in units (U) rather than cubic centimeters. Many of these syringes have 2 calibrations (U20 U40 or U40 U80) and it is important to have the patient thoroughly understand which scale he is using. It is advisable however to employ a syringe having one calibration only. Special syringes are available for blind diabetic patients.
- 3 **Sites of injection** Insulin is usually administered subcutaneously. The site of injection is generally the anterior thigh but insulin may also be given in the lateral thigh, in the arms or anterior abdomen or in unusual circumstances subcutaneously in another part of the body. It is important that the site be constantly changed so that the same site is not injected more often than once every 2-3 weeks. Crystallin and regular insulin may be administered I.V. to patients who have been taking insulin with an allergic reaction. Do not give PZI or PTH intravenously.

#### Diet in Diabetes

The nutritional needs of the diabetic patient are not significantly different from those of normal individuals. The principal question to be settled is the quantity and type of carbohydrate to be allowed in the diet. (For detailed food charts in making up diets see pp 44-51 and for examples of diabetic diets see p 55.)

N 1. Whenever possible diabetic diets should be made up in terms of household measures rather than weight for clinically the extra accuracy gained by weighing is generally unnecessary.

The following factors must be taken into consideration in estimating the diet:

- A Calorie Needs (See p 45.) The caloric needs of the diabetic are similar to those for a non-diabetic individual and the same variables must be considered. In general one should remember that the diabetic patient should be kept at normal or slightly subnormal weight levels and never permitted to become obese.
- B Protein. Protein must be adequate and high protein diets reduce desirable because the available glucose (50%) from protein is released more slowly for utilization than ingested carbohydrate. At least 1 Gm. of protein per Kg. (0.45 Gm./lb.) of body weight should be given although 1.2-2 Gm./Kg. (0.7-0.9 Gm./lb.) are preferable.
- C Carbohydrate. Carbohydrate should not be given in concentrated form. Preference should be given to 3% and 10% fruits and vegetables. These take longer to digest and absorb and a less variable blood sugar level is obtained. The question of adequate versus restricted carbohydrate in the diet is still unsettled. In general the view is taken that in the diabetic the aim is to keep the individual as close to physiological normal as possible and hence to keep his carbohydrate at approximately normal levels and to administer insulin if necessary to control a resulting hyperglycemia and glycosuria. In general therefore 3-3 Gm. of carbohydrate per Kg. (0.8-1.4 Gm./lb.) of body weight is recommended at the start of treatment. If patient's tolerance increases with treatment gradually increase carbohydrate to 4 Gm. per Kg. (1.8 Gm./lb.) of body weight. This is a general rule and in some mild diabetes it



may be advisable to keep the carbohydrate level down to avoid the use of insulin. However, both for physiological and for psychological reasons, the carbohydrate level should in no case be below 100 Gm. per day.

- D Fat. After the carbohydrate and protein amounts have been determined, fat is given to make up the remaining caloric requirements.
- E Vitamins. Patients with diabetes tend to develop vitamin deficiencies, especially of the B complex. The reasons are not always clear but may be due to inadequate food intake, restricted diets, or increased requirements or improper utilization of vitamins. Deficiencies on adequate diets are rare. If they occur, treat as needed (see p. 58).
- F Frequency of Feeding. Diabetics should be given small frequent feedings rather than large meals. By frequent feedings, the use of high protein intake and less concentrated carbohydrate foods, one can maintain a lower and more even blood sugar level with less glycosuria. An excellent plan is to divide the feedings into six meals: three regular meals and three small feedings (e.g., milk) at mid morning, mid afternoon, and bedtime.

#### Other Factors Influencing Diabetes

- A Exercise. Exercise enhances the oxidation of sugar, hence it diminishes the need for insulin. Therefore, exercise in moderation is beneficial. However, patients taking insulin should be cautioned against strenuous exercise without fortifying themselves previously with extra carbohydrate. (It is not uncommon to have a hypoglycemic reaction after a set of tennis.) When regulating a patient, have him perform approximately the same amount of exercise as will be required by his normal activities. This is true also of hospital-regulated diabetics.
- B Complicating Factors. A large number of factors adversely affect the course of the patient with diabetes. All of these conditions operate by altering the absorption of glucose, by interfering with carbohydrate oxidation, or by causing excessive carbohydrate formation. The most important of these factors are infections, especially those of pyogenic nature with fever and toxemia. Any infection is serious in a diabetic, for it completely upsets the equilibrium established by therapy, always increases the need for insulin, and is one of the most common precipitating causes of ketosis and acidosis. Therefore, any and all infections in the diabetic are to be avoided whenever possible; when they occur, they must be treated promptly and vigorously. During severe infections, it is generally advisable to discontinue PZI and to begin therapy in divided doses: 6 times daily with regular or crystalline insulin as needed to cover postprandial glycosuria.
- C General Factors. Patients with diabetes should live as nearly normal hygienic lives as possible. They should be assured of adequate rest, should be able to eat at home if at all possible, and should engage in an occupation requiring at least moderate exercise but must avoid strenuous occupations of greatest importance. They should have a good general knowledge of diabetes.

## STEPS IN THE MANAGEMENT OF THE DIABETIC PATIENT

There are many adequate methods for managing diabetics. The following is a plan suggested by the author which is felt to be practical and physiologically sound.

### TYPE 1 - DIABETIC CRASH-UP

1. Complete history and physical examination for diagnosis and to rule out the presence of any coexisting or complicating disease.
2. Urinary  $\text{HbA}_{1c}$  for qualitative sugar on a morning fasting urine specimen and on specimens collected 2-3 hours after each meal. If sugar is present check fasting and diastolic blood sugar.
3. Blood sugar examination. Fasting and 2-hour postprandial levels are determined or if necessary a glucose tolerance test is performed. In elderly patients or in the presence of renal disease it is advisable to perform a glucose tolerance test with simultaneous urine glucose to determine the approximate renal threshold. If this is very high (over 180-180 mg %) it may be necessary to use blood sugar levels as a check on adequacy of therapy rather than the glycosuria.

### TYPE 2 - CALCULATING THE DIABETIC DIET (see p. 35) for examples of diabetic diets

1. Determine the caloric needs of the patient. This is the same as for the non-diabetic (see p. 43).
2. Calculate the protein in CHO and fat content of the diet as outlined in the diabetic diet section on pp. 47-50.
3. Divide the diet into the following:
  - a. The main meal. The meal is as desirable as possible (i.e., a early breakfast and late dinner). This will provide the absorption of glucose over a long period of the day.
  - b. The small feedings to be taken between meals and at bedtime. Milk and low CHO fruits are preferred for this.

### TYPE 3 - THE DIETARY MANAGEMENT OF THE DIABETIC PATIENT

1. Determination of amount of glycemia. Have patient eat his diabetic diet for 1 day preferably without  $\text{HbA}_{1c}$  activity. For the first 24 hours he is to collect and label fractional excretions of urine (Patient voids just before breakfast and dinner, and at bedtime).
  - a. Urine No. 1. All urine voided from breakfast to just before lunch. This is pooled and a few drops taken for qualitative sugar. The remainder is voided.
  - b. Urine No. 2. All urine from lunch to just before dinner. Pool and analyze as above.
  - c. Urine No. 3. All urine from dinner to just before retiring. Pool and analyze as above.
  - d. Urine No. 4. All urine from retiring to just before breakfast. Pool and analyze as above.
- The few drops of each individual urine fraction are analyzed qualitatively for sugar and the remainder pooled for the daily total quantitative sugar.

- 2 Calculation of approximate insulin requirements from quantitative urine sugar determinations. Since roughly 1 unit of insulin will cover 2 Gm. of glucose the insulin needs in the uncomplicated diabetic can be calculated as follows

Gm. of Glucose in 24 hour Urine Specimen	Approximate No. of Units of Insulin Needed per 24 Hours
2	

The insulin (24 hour requirement) is generally given as NPH or as a mixture in a single dose  $\frac{1}{2}$  hour before breakfast. The usual mixtures are 2:1 or 3:1 (crystalline zinc PZI) or NPH: regular mixtures.

- a In severe or complicated diabetes because the patient needs insulin immediately these measures cannot be performed (see # 403)
  - b High renal threshold. In certain elderly patients or those with renal disease who have a high renal threshold for sugar this method will be without value. These patients must be controlled by the determination of the blood sugar levels while fasting and 1 hour after meals. In these cases begin with small doses of long acting insulin (5-10 units/day) and increase as indicated by tests.
- 3 Adjustment of insulin dosage and mixture. The patient continues to collect his urine fractions as outlined above and the dosage and composition of the insulin mixture is determined each morning after completing the qualitative sugar analysis for the previous day. Quantitative sugars are usually not necessary after the first day. The amount and time of glycosuria on the preceding day determines the readjustment to be made. The glycosuria at any time must be kept at a minimum (no greater than green reduction in any specimen). In general especially with longer acting insulins changes should not be made frequently simply because occasionally marked insulin reactions occur.
- a If all specimens are green no adjustment of dosage or composition of insulin is necessary.
  - b If glycosuria (greater than green reduction) occurs after breakfast or after the noon meal the regular insulin is increased.
  - c If glycosuria (greater than green reduction) occurs in the afternoon after the evening meal or before breakfast the protamine zinc insulin is increased.
  - d If glycosuria (greater than green reduction) occurs in all specimens both regular and protamine zinc insulins are increased.
  - e Amount of increase of insulin will vary with each patient. Generally a very rough guide for the increase of insulin is as follows:
    - (1) Yellow reduction: Add up to 3 units
    - (2) Orange reduction: Add 5-10 units
    - (3) Brick red reduction: Add 10-15 units
  - f If there is no glycosuria (specimen remains blue) the patient should be questioned for evidence of hypoglycemia and each urine voided should be examined. Adjustment of dosage must be made in accordance with the findings.

- 4 Readjustment of the size of feedings. If variations of the insulin dosage and composition do not maintain the glycosuria at a minimum for a given period, the dietary intake for the preceding meal should be decreased and the intake for other meals increased a like amount.

#### STEP D - FOLLOW-UP OF PATIENT

After patient has been adequately controlled, he should be seen at regular intervals arranged as needed to check for any change in the patient or in his diabetic status.

- 1 Hypoglycemic reactions. Carefully question patient as to occurrence of any hypoglycemic reactions. If these occur, lower insulin according to time of day they take place.
- 2 Examine patient's urine. If all urine is entirely free of sugar, the patient is controlled (if renal threshold normal) be careful of hypoglycemic reactions, however, if all urines are blood-early in therapy, for patient's tolerance will improve under therapy. There is no contraindication to having some glycosuria. (On the contrary, there is some evidence that moderate glycosuria per se is not harmful if the metabolic needs of the body are being fulfilled. However, it is usually best to keep a quick control over patient as they tend to ignore their disease entirely.) If there is marked glycosuria in any urine, the insulin is adjusted accordingly.
- 3 Weigh patient. Follow patient's weight to be sure that the weight is increasing and decreasing as remaining stationary as desired. If not, alter the diet accordingly.
- 4 Draw blood for fasting blood sugar level to determine whether fasting hyperglycemia is being adequately controlled (This need not be done on every visit; in fact, it can be done quite infrequently once the patient is standardized).

## COMPLICATIONS OF INSULIN THERAPY

### HYPOGLYCEMIA (code No 574)

Hypoglycemia is the most common complication of insulin therapy and usually occurs when the diabetic fails to eat the proper amount of food. It is manifested by weakness, hunger, irritability, tremor, and tremors and convulsions. If of which are relieved promptly by the administration of glucose. If a diabetic patient is unconscious and if diagnosis of coma or loss of reaction is impossible or in doubt, give 50% glucose IV. This will usually overcome the situation and will not generally harm the patient in diabetic coma.

#### Prevention

- A. Education. Because of the danger of a low reaction, the diabetic patient should carry a small supply of sugar or glucose. If he feels the onset of a reaction, he should take some sugar.
- B. Insulin Card. Every diabetic should carry a card with the following information:

## I AM A DIABETIC AND TAKE INSULIN

If I am behaving peculiarly give me sugar or hard candy or orange juice slowly. If I am unconscious call an ambulance immediately take me to a physician or a hospital and notify my physician. I am not intoxicated.

My Name is \_\_\_\_\_

Address \_\_\_\_\_ Telephone \_\_\_\_\_

Physician's Name \_\_\_\_\_

Physician's Address \_\_\_\_\_ Telephone \_\_\_\_\_

### Treatment

- A Mild Hypoglycemia If patient is conscious and able to swallow sugar, glucose or orange juice may be given.
- B Moderate to Severe Hypoglycemia Do not attempt to feed patient if unconscious. If patient is unconscious one of three methods may be used:
  - 1 I V glucose (treatment of choice) 50 cc (5 12 dr) of 50% glucose I V slowly. As soon as consciousness is restored oral feedings may begin.
  - 2 Epinephrin, adrenaline. If patient is well nourished especially if using short acting insulin and liver is not depleted of glycogen epinephrine 0.5 to 1.0 cc (8 15 m) of 1:1000 solution subcut may cause return of consciousness so that food may be taken by mouth.
  - 3 Rectal feeding. If patient is unconscious and I V glucose is not available (and if epinephrine is either not available or not feasible or successful) glucose by rectum may be life saving. Add 2 Tbsp of syrup or honey to a pint of warm water and give slowly by rectum.
- C Prevention of Relapse When patients taking protamine zinc insulin develop reactions they should be carefully watched for danger of relapse. High protein food such as milk should be given in addition to carbohydrate.

## OTHER COMPLICATIONS OF INSULIN THERAPY

### Allergic Reaction

Fortunately allergic reactions are very rare and most reactions are localized. These individuals are generally sensitive to pork pancreas from which about 60% of commercial insulin is made (other 40% from beef). These patients should be given pure beef insulin preparation (Special Insulin) which is put up in 10 cc ampules of U40. If patient is still sensitive desensitization measures should be tried (see p. 113).

### Lipostrophy

This rare complication consists of atrophy of subcutaneous fat at the sites of injection. This may be caused by improper rotation of injection sites but some cases occur in spite of careful therapy.

This patient should use U50 or U100 insulin estate injection site and make injection at body areas which are clothed at all times

## COMPLICATIONS OF DIABETES

### CHRONIC COMPLICATIONS

There are certain disease processes that tend to occur with greater frequency in diabetic patients than in non-diabetics and a few conditions that are rather typically associated with diabetes. They are mentioned here in order to call attention to them. Their therapy is generally that of adequate control of the diabetes and the abey of the coexisting or underlying disease. The most common diseases are

- A Arteriosclerosis Especially of the peripheral arteries of the legs. For therapy see p 308
- B Diabetic Peripheral Neuritis (See p 358)
- C Diabetic Ocular Complications Incl cataract which is treated surgically and retinitis for which no form of therapy is of any avail
- D Renal Complications Inter-capillary glomerulosclerosis characterized by hypertension, albuminuria and edema. Treat as for glomerulonephritis (see p 293)

### ACUTE COMPLICATIONS OF DIABETES

When the amount of insulin in the body is inadequate freedom is held, abnormal metabolism results with ketone body formation and finally with acidosis. Infection which causes an increased demand for insulin usually precipitates ketosis. There is an early mild phase and a late or severe one.

- A Diabetic Illness Without Acidosis  $\text{CO}_2$  combining power  $\geq$  normal, slightly depressed (above 50-60 Vol % or 27 mEq)
- B Diabetic Acidosis Reduction of  $\text{CO}_2$  combining power (below 50 Vol % or 27 mEq). The patient may be one hour pre-comatose or comatose.

### DIABETIC KETOSIS (Without Acidosis) (code No 543)

In this disorder ketone bodies are found in the urine and their presence is the best diagnosis. Examine the patient if infection or other precipitating factor. The fluid and electrolyte balance is unaltered.

#### Treatment

Patient should be hospitalized for regulation if ketosis is severe.

- A Treat any infection which may aggravate the disorder and metabolic
- B Feeding Arrange diet to consist of equal feedings with interval feedings between each meal and in the evening.

**■ Insulin**

- 1 If ketosis is very severe use only short-acting insulin. Give insulin to cover each meal as necessary until the urine is free from ketone bodies. Then begin reducing insulin dosage slowly as tolerance to carbohydrate improves.
- 2 If ketosis is not severe treat and regulate as uncomplicated diabetes.

**D Follow up** When ketonuria has cleared patient is managed as for uncomplicated diabetes according to the severity of his disease (see p. 399)

## DIABETIC ACIDOSIS (Diabetic Coma) (code No. 543)

When ketone formation is proceeding at a rapid rate the fluid and electrolyte balance and pH of the body are upset (see below). The ketone bodies are organic acids which replace the  $\text{HCO}_3^-$  in the body and also are excreted from the body combined with fixed base. This loss of fixed base and the disturbance of the buffering systems leads to acidosis. The increase in the glucose in the blood produces diuresis of needed body fluids.

**Diagnosis**

Diabetic acidosis is manifested by headache, irritability, drowsiness, hyperpnea and fever. Nausea, vomiting, diarrhea and abdominal pain may also be present. The sweetish, fruity acetone breath may be detected. On physical examination the skin and mucous membranes are usually dry, blood pressure low, eyeballs soft and pulse usually rapid and thready.

**Principles of Therapy**

For emergency management see below.

The principles of therapy whether the patient is precomatose or in coma are the same. It is imperative that a patient in acidosis be hospitalized and treated as a medical emergency. Each case must be individualized.

- A Insulin** in large amounts is necessary to bring about a return to normal metabolism. Use short-acting insulin; never treat patients in coma with PZI. The first dose of insulin should be 100-200 units; one half should be given I.V. and the other half subcutaneously. Insulin may also be added to I.V. fluids being administered. Because of the mode of action of insulin (see p. 393) there is no need to repeat sooner than in 1-2 hours. The dose may then be repeated subcut. or I.V. giving 50-75 units every 1-2 hours as needed until the ketonuria begins to disappear. If shock is present the insulin should be given I.V. because of the unreliable absorption during shock of material given subcutaneously.
- B Glucose.** In diabetic acidosis one is treating the ketosis and acidosis and not the hyperglycemia and glycosuria. Although the patient with acidosis may have a high blood sugar level the total available carbohydrate stores may actually be very low. Therefore since it is necessary to have an adequate glucose supply upon which insulin can act in overcoming acidosis these

patients should be given glucose when the blood sugar level has begun to fall rapidly. It has been shown that ketosis can be reduced by giving very large amounts of glucose to diabetic patients who are deprived of insulin. The sooner the normal metabolic pathways are reestablished the sooner the excess fat oxidation ceases and ketonemia is overcome. In addition it is possible to precipitate hypoglycemic reaction in a patient with low sugar reserves before the ketosis is brought under control.

- C Fructose and Inulin Sugar. It has been shown that after I.V. infusions fructose disappears from the blood stream of diabetics as rapidly as it does from normals. It has been suggested therefore that this sugar be substituted for glucose in the treatment of diabetics because it is utilized in the absence of insulin. However, there is some evidence to show that in spite of its utilization in the diabetic it has no antiketogenic effect without insulin. Until this critical question is settled one should continue to use glucose and insulin in the management of diabetic acidosis.

D Fluids and Electrolytes

1. Fluids must be given to replace those lost by diarrhea and vomiting. These are usually best given I.V.
2. Adequate sodium chloride is very important. This replaces fixed base in the extracellular fluid and so helps in overcoming the acidosis. As a result of ketosis the loss of sodium chloride from the body may be as great as 30 Gm (10% of average total body sodium) in 24-48 hours. In the mild case sodium chloride needs to be replaced and sodium chloride solution with glucose is usually adequate fluid therapy.
3. Replacement of bicarbonate buffer. As the ketone bodies are excreted or oxidized  $\text{CO}_2$  is formed which replaces the disappearing ketones and the  $\text{CO}_2$  combining power returns to normal. However, in patients with severe uncomplicated metabolic acidosis it may be advisable to administer more rapidly available  $\text{HCO}_3^-$  and fixed base (i.e.  $\text{Na}^+$ ). This may be given I.V. as sodium bicarbonate or M/6 sodium lactate.
4. Potassium replacement. As sodium is administered (as sodium chloride, sodium bicarbonate or sodium lactate) and glucose is metabolized and stored the potassium which has entered the extracellular fluid migrates rapidly intracellularly or is washed out with the fluid through the kidneys. When this occurs there may be a temporary and dangerous extracellular potassium deficiency with weakness, respiratory distress and at times cardiac arrest. Solutions containing potassium must be given to offset this and generally when I.V. glucose becomes indicated potassium may be added to the infusion mixture (see p. 23). It must be used with extreme caution in the absence of adequate urinary output. The level may roughly be checked with the E.K.G. (p. 18).

Treatment

- A. Emergency Measures. The following is an outline of therapy that may be required in the average patient in diabetic coma.



however each case must be individualized and therapy modified as necessary according to the needs of the patient

- 1 Hospitalize patient Keep patient warm avoid excessive warmth Avoid the use of barbiturates and narcotics
- 2 If in D/DCCF treat with I V plasma and other shock measures especially vasopressors (see p 31)
- 3 Blood chemistry Draw blood for  $\text{CO}_2$  combining power and blood sugar also for sodium potassium and chloride If these tests can be performed
- 4 Give insulin at once
  - a Through same needle used for drawing blood give 100 units of regular or crystalline insulin I V immediately as well as a like amount subcutaneously
  - b Repeat insulin giving 50-75 units subcut every 1-2 hours until there is rapid diminution in blood or urinary sugar
- 5 Catheterize patient An indwelling catheter may be left in place allow this to drain continuously Examine spot (periodic) urine specimen every hour for ketone bodies and sugar
- 6 Fluids electrolytes and glucose
  - a Begin I V infusion of saline May also begin clysis of saline M/6 sodium lactate or other indicated solutions at same time (see p 21) As soon as urinary sugar has changed to olive or green reduction change I V fluids to 5% glucose in saline to which is added  $\frac{1}{2}$  1 unit of insulin per gram of sugar (25-50 units insulin per liter) and 20 mEq potassium and possibly phosphate The urine should contain sugar at all times to avoid hypoglycemic reactions
  - b As soon as reports come from laboratory if  $\text{CO}_2$  combining power is below 5 mEq /liter (10 Vol %) calculate amount of sodium lactate or sodium bicarbonate desired (see p 21) and administer immediately (To administer sodium bicarbonate I V merely dissolve chemically pure sodium bicarbonate in 200-300 cc cool distilled water and administer Do not heat or sterilize the solution)
  - c Gastric lavage may be performed with introduction of 200 cc of physiological saline or 5%  $\text{NaHCO}_3$
  - d As long as patient is unconscious administer 5% glucose in saline or other salt solution as indicated (about 80 drops per minute) See p 405
  - e As soon as patient is conscious and able to swallow give fruit juice (200 cc orange juice with 1 tablespoon honey syrup or glucose) every 3-4 hours until keturia has stopped Stop I V glucose and fluids

## B Follow up

- 1 Potassium deficiency After 4 to 8 hours of administration of I V fluids watch patient carefully for potassium deficiency (i.e. weakness respiratory distress) and check the Ecg (see p 19) Give solutions containing potassium (see p 25) as indicated It may be advisable to begin administration of potassium as soon as the maintenance is begun but this is still not settled When patient is able to swallow give supplementary potassium salts by mouth as this is the safest route

- 2 Oral feedings and fluids. If keto uria is developing or is rapidly improving (usually in 24-48 hours) and the patient is conscious the following may be given:
  - a Small frequent feedings of liquid and semi liquid foods containing 75 Gm glucose and protein (as milk) every 3-4 hours day and night and cover with 25-35 units regular insulin every 4 hours
  - b Fluids by mouth
  - c Examine urine for sugar and ketone bodies every 3-4 hours
- 3 Regular diet. After 24-48 hours if patient shows satisfactory improvement place on regular diet and begin regulation as outlined on p 399

## DIABETES ASSOCIATED WITH OTHER CONDITIONS

### PREGNANCY

The management of the pregnant diabetic is little different from that of any other diabetic.

- A During the early period of pregnancy there is often a lowering of the renal threshold and considerable lability of the blood sugar level.
- B During the latter three months there is often a marked decrease in tolerance necessitating increased insulin dosage. This is of minor importance however and may go through pregnancy without significant changes in tolerance.
- C Before the onset of labor and delivery it is advisable to change to short acting insulin to avoid possible reaction from lack of food.
- D In view of work suggesting sex hormonal imbalance in pregnant diabetes therapy with estrogen or progestone or both has been advocated as being of value in diminishing fetal mortality. However carefully controlled studies using modern diabetic treatment methods show as good or better results without resorting to this expensive and troublesome procedure.
- E Since many diabetic pregnancies go beyond expected term or the infants are unusually large it has been suggested by many that pregnancy be terminated at about 36 weeks. The preferred method appears to be cesarean section.

### The Care of the Infant

The infant of diabetic mother is a premature. Keep infant in incubator under oxygen if first several days. Observe the newly born infant carefully for the first 72 hours for hypoglycemic reactions that may occur as a result of fetal cell hyperplasia. This is more apt to occur in the newborn of poorly controlled diabetics.

## SURGERY

Surgery in the diabetic at present presents little hazard over that of the surgical procedure per se. However, there are certain problems peculiar to the diabetic and these problems naturally vary with the severity of the disease and the urgency of surgery.

### Emergency Surgery

- A For Non Traumatic Conditions** Usually diabetics requiring emergency surgery for non traumatic disorders are in ketosis with or without acidosis and require immediate treatment of their diabetes. These patients should be treated as patients with acidosis or coma (the latter if a general anesthesia is to be used). The general program should be as follows:
- 1 Draw blood for  $\text{CO}_2$  combining power and blood sugar also for electrolyte panel (sodium potassium chloride) if possible
  - 2 Begin 5% glucose in saline infusion I V slowly (not over 70 drops per minute) and continue infusion throughout surgical procedure. One unit of insulin per 2 Gm glucose may be added to the infusion (25 units for each 1000 cc of 5% glucose)
  - 3 Give 50 units short acting insulin I V if ketosis is present
  - 4 After returning from surgery continue therapy as for diabetic coma (see p. 404) until oral feeding can begin and ketosis and hyperglycemia are controlled
- B For Trauma** Although increased carbohydrate tolerance develops rapidly as a result of trauma, the principle danger in a treated diabetic who is injured is the possibility of having a severe hypoglycemic reaction because he fails to eat. Therefore, if the patient is conscious, give sweetened orange juice or candy by mouth. If surgery is necessary, give 5% glucose I V in water or saline slowly. One may add 1 unit insulin per 2-3 Gm glucose to the infusion; however, the need is not so much for insulin as for glucose to avoid hypoglycemia. When surgery has been completed, treat according to severity of disease (see p. 394).

### Elective Surgery

#### A Initial Hospital Measures

- 1 Patient should enter hospital several days before surgery
- 2 Discontinue protamine zinc insulin
- 3 The diabetes should be brought under optimum control with regular or crystalline insulin
- 4 There should be no ketosis

#### B During and After Surgery

- 1 No food or insulin should be administered on the morning of surgery
- 2 Management during surgery
  - a If the patient's diabetes is mild and has been properly controlled. If he does not tend to develop ketosis and if the surgery is not too extensive, he may be operated on without food or insulin
  - b If the patient's diabetes is moderate or severe or if extensive surgery must be performed, begin infusion of 5%

glucose in line or water to which has been added 1 unit regular or crystalline insulin per 2 Gm glucose. Continue infusion throughout surgical procedure. Give insulin at about 60-70 drops per minute.

- 3 After surgery patient should have small frequent feedings (50-75 Gm carbohydrate) every 3-4 hours covered with 15-25 units of crystalline insulin subcutaneously before the meal. These small feedings are continued until normal nutrition can be re-established.
- 4 If gastrointestinal surgery has been performed and patient cannot take food by mouth, nutrition can best be maintained by parenteral methods: give 1000 cc 5% glucose in 5% amino acid solution I.V. always over a period of 4 hours. This should be covered with 15-40 units of crystalline insulin before beginning infusion. Three liters per day is an average requirement. This therapy may be continued until oral nutrition can be resumed.

### HYPERINSULINISM

(Adenoma code No 871 8044A)

(Without Tumor code No 871 784)

Hyperinsulinism is caused by a excessive production of insulin and manifested by attacks of weakness, hunger, irritability, faintness, and tremors and convulsions, all of which usually occur on an empty stomach long after meals and are relieved promptly by the administration of glucose. Hypoglycemia of prolonged episodes usually below 50 mg %. A glucose tolerance curve dropped up to the exceedingly low levels of only after 5-6 hours is characteristic. Hypoglycemia of hepatic, nervous or other endocrine origin must be ruled out to establish the diagnosis.

### Treatment

A. Symptomatic Treatment. As for hypoglycemic reaction from insulin over dosage (see p 402).

### B. General Management

- 1 Control of pain (ACTH). The administration of ACTH (its hyperglycemic effect) has been shown to be of considerable benefit in the management of some children suffering from this condition. Some children without adequate reason to have been acutely malnourished or treated intermittently with this drug.
- 2 Diet: High protein, high calorie, high fat, low-carbohydrate.
  - a. The diet is low in carbohydrate in order to avoid stimulation of the pancreas to elaborate insulin. Rapidly titrated carbohydrate or replaced by slow acting ones (e.g. 3-10% vegetable oils and fruits and bananas and prunes). Fats in this important source of a slowly released carbohydrate which apparently has less stimulating effect on the pancreas and is useful to a poorly fed child.
  - b. Small feedings. The diet is best divided into six or more meals a day. It may be necessary to feed the patient at

regular intervals throughout the entire 24 hours. If the hypoglycemia is as severe as this, it is advisable not to prolong medical therapy but to prepare the patient properly for surgery.

- 3 Sedation. Phenobarbital phenobarbitone 15-20 mg ( $\frac{1}{4}$  -  $\frac{1}{2}$  gr) q i d may be valuable in reducing neuromuscular irritability.
- 4 Restriction of physical activity. Exercise increases utilization of glucose, thereby exaggerating the effect of excess insulin. If exercise is unavoidable, such activity should be preceded by supplementary carbohydrates.
- 5 Identification card. Patient should carry a bracelet or card similar to that used by a diabetic (see p. 40).
- 6 Emergency CHO. Patient should be required to carry a small supply of rapidly available carbohydrate (candy lumps of sugar) at all times. He is to avoid taking these except when definitely indicated.
- 7 Surgery. Complete excision of hyperplastic or adenomatous islet tissue when this is found to be the cause.

## Chapter 15

# HORMONES AND HORMONE LIKE AGENTS

## PITUITARY AND PITUITARY LIKE HORMONES

- The pituitary consists of two parts  
 A Posterior pituitary which is devoid of direct neural innervation  
 B Anterior pituitary which is directly innervated by the hypothalamus  
 C Pituitary like hormones that influence the gonads are secreted by the placenta during pregnancy

## ANTERIOR PITUITARY HORMONES

All of the anterior pituitary hormones are polypeptides. Some are secreted by the hypothalamus and some by the anterior pituitary. The hypothalamus secretes the releasing and inhibiting hormones which act on the anterior pituitary. The anterior pituitary secretes the growth hormone (GH), prolactin (PRL), thyroid stimulating hormone (TSH), follicle stimulating hormone (FSH), and luteinizing hormone (LH). GH is the most abundant and is secreted by the somatotrophic cells. PRL is secreted by the lactotrophic cells. TSH, FSH, and LH are secreted by the gonadotrophic cells. GH is secreted in pulses and its secretion is under the control of growth hormone releasing hormone (GHRH) and growth hormone inhibiting hormone (GHRIH). PRL secretion is under the control of prolactin releasing hormone (PRH) and prolactin inhibiting hormone (PIH). TSH, FSH, and LH secretion is under the control of gonadotropin releasing hormone (GnRH) and gonadotropin inhibiting hormone (GnIH).

## Corticotropin (ACTH)

Corticotropin (ACTH) has been shown to have a stimulatory effect on the secretion of the adrenal cortex. It is secreted by the corticotrophic cells of the anterior pituitary. ACTH is a polypeptide hormone with a molecular weight of approximately 4500. It is secreted in pulses and its secretion is under the control of corticotropin releasing hormone (CRH) and corticotropin inhibiting hormone (CIH). ACTH has a stimulatory effect on the secretion of the adrenal cortex, which secretes the glucocorticoids and mineralocorticoids. ACTH also has a stimulatory effect on the secretion of the adrenal medulla, which secretes the catecholamines. ACTH is secreted by the anterior pituitary and its secretion is under the control of the hypothalamus.

## ACTH

1. ACTH is a polypeptide hormone secreted by the anterior pituitary.
2. ACTH has a stimulatory effect on the secretion of the adrenal cortex.
3. ACTH has a stimulatory effect on the secretion of the adrenal medulla.
4. ACTH secretion is under the control of the hypothalamus.

## 412 Pituitary Hormones

- 5 Increased urinary  $\Delta^4$  ketosteroids and 11 oxysteroids
- 6 Fall in circulating eosinophils and lymphocytes and elevation of polymorphonuclears

B For clinical effects uses and dosages see page 423

### Pituitary Growth Hormone (PGH)

Pure PGH has been employed in normal humans pituitary dwarfs and panhypopituitary individuals. In no case has there been any evidence of growth as measured either by reticoblastic effect or actual physical growth. The older crude growth hormone preparations have likewise been of no benefit under controlled experimental conditions.

### Lactogenic Hormone

Has not been employed in human research. Its presence is necessary for the initiation and apparently for the continuation of lactation in breasts which have been prepared for lactation by estrogens and progesterones during pregnancy.

### Follicle Stimulating Hormone (FSH)

FSH has different actions in male and female. In the female FSH stimulates the development of ovarian follicles. In the male it stimulates the germinal epithelium of the testis to produce spermatozoa. It apparently has no effect on the Leydig cells hence does not influence testosterone secretion. Pure FSH has not been used clinically but in cases of hypogonadotropic eunuchoidism a purified preparation has been employed after initial stimulation of Leydig cells with chorionic gonadotropins to initiate spermatogenesis (see page 388). At present no good FSH preparation is commercially available.

### Interstitial Cell-Stimulating Hormone (ICSH) (Luteinizing Hormone)

A In the female ICSH apparently has a dual action

- 1 Stimulates growth of theca cells
- 2 Transforms the mature follicles into corpora lutea

B In the male it stimulates the Leydig cells of the testis with resultant testosterone secretion.

There is no good commercial pituitary ICSH. Clinically ICSH is substituted for by use of chorionic gonadotropins which have a similar action (see page 413).

### Thyroid Stimulating Hormone (TSH)

TSH is exceedingly efficient in stimulating the thyroid gland. It has limited clinical usefulness. At present its principal uses are to differentiate pituitary hypothyroidism from primary hypothyroidism. It has also been used in an attempt to stimulate metastatic thyroid cancer to take up radiolodine for therapeutic purposes.

Recently it has been advocated for treatment of thyroiditis but its place in the management of this disease is still open to question.

Dosage 10-25 mg every 8 hrs for 3 days. Repeat  $^{131}$ I uptake. If uptake increased primary hypothyroidism is not present.

### Other Hormones

The pituitary probably elaborates other hormones (e.g. luteotropin) but their exact physiological roles are not known at present.

## POSTERIOR PITUITARY HORMONES

The posterior pituitary hormones are peptide compounds of 8 amino acids. Their exact chemical structures have been determined and they have recently been synthesized. Like the anterior pituitary hormones they are effective only when administered parenterally (give I.M.). They exert the following actions:

1. They raise blood pressure (pressor action) (anesthetized animals)
2. Cause fluid retention without osmotically equivalent sodium retention (antidiuretic action)
3. Cause uterine contractions (oxytocic action)

To date there has not been a separation of the antidiuretic from the pressor principle. They may be identical. The oxytocic action may likewise have some pressor effect.

### CLINICAL

- A. Antidiuretic. Pressor principle is used primarily for the treatment of diabetes insipidus, also to prevent and control abdominal distention (For Diabetes Insipidus see page 388)
- B. Oxytocin is employed in obstetrics when indications for the induction of uterine contraction exist.

### Preparations Available

Name	Action	How Supplied	Average Dosage
Vasopressin Tannate Injection, N.N.R. (Pituitrin Tannate)	Antidiuretic pressor	Only solution 5 units/cc	0.3-1 cc (3-16 u) q 12-72 hr
Isopressin Injection U.S.P.		Aqueous solution 20 units/cc	0.2-0.5 (4-8 u) q 2-4 h
Oxytocin Injection U.S.M. (Pitocin®)	Oxytocic	Solution 10 units/cc	0.3-1 cc (3-16 u) as indicated

## PITUITARY LIKE HORMONES ELABORATED BY PLACENTA

The most important of the pituitary like hormones is that elaborated by the placenta during pregnancy. The hormone is referred to as chorionic gonadotropin. It physiologically acts on almost identical with that of FSH above. It has recently been shown that this hormone appears to exert its actions only if an intact anterior pituitary gland is present. It is of no value in inducing spermatogenesis or ovulation or maintaining corpus luteum by itself. Many of its alleged effects have been due to the presence of FSH whose action the presence of chorionic gonadotropin may potentiate.

### Clinical Indications

1. Cryptorchidism. In a few selected cases chorionic gonadotropin may induce descent of the testis.
2. Hypogonadism. Chorionic gonadotropin is useful in some



## 414 Thyroid Hormone

types of hypogonadism although testosterone medication is generally preferred

- B In the Female Chorionic gonadotropin may aid in inducing ovulation and maintaining corpus luteum in a few selected cases of sterility if adequate FSH is present

### Preparations Available

- A Chorionic Gonadotropin N N R is derived from the urine of pregnant women is available commercially under a wide variety of trade names It is marketed in ampules of 100 500 1000 and 5000 I U per cc
- B Equine gonadotropins derived from the serum of pregnant mares is also available commercially This is a mixture of FSH and ICSH It is not generally recommended because of its marked sensitizing effect and production of antihormones by protracted use Only short courses should be employed

### Average Doses

Usual doses range from 200 1000 units every day or every other day

## THYROID HORMONE

The active principle of the thyroid gland appears to be the iodine containing amino acid thyroxine Thyroxine probably never occurs in the free state in the organism but is contained in a protein molecule thyroglobulin Another iodine containing amino acid diiodotyrosine with weaker physiological effects is also found in the gland Recently tri iodothyronine has been isolated from the thyroid It is about 4 times as potent as thyroxine and acts more rapidly Its exact physiological role is unknown The action of the thyroid hormone is that of a general cellular metabolic stimulant with resultant increased oxygen consumption (i.e. increased metabolic rate) Its exact mode of action is unknown

### Method of Administration

Thyroid hormone either in the form of thyroglobulin (desiccated thyroid) or thyroxine is effective when taken orally Little effect is noted after a single dose for about 24 hours and the maximal effect is not reached for 6 10 days After thyroid medication is stopped there is a slow loss of the effect depending on the initial B.M.B. and the level reached during thyroid medication In general it may be stated that at least 6 weeks must elapse after thyroid medication has been discontinued before one can be reasonably certain that the major thyroid effects have worn off

### Clinical Uses

Thyroid hormone is indicated only in thyroid deficiency states Its use as a general metabolic stimulant is not indicated and is worthless It has been shown that patients with thyroid deficiencies rarely require over 0.13 Gm (2 gr.) of Thyroid U.S.P. daily Patients without deficiency states can easily tolerate 0.3 to 0.5 Gm (5 to 7½ gr.) or more daily without any effect on B.M.B. or other metabolic

A good general rule is that if a patient requires

over 23 gr of Thyroid U.S.P. daily his need for thyroid medication should be questioned

Preparations Available

**A Thyroid U.S.P.** **B** (also called thyroid) This is the preparation of the thyroid gland. It is a mixture of the thyroid gland and contains the same amount of iodine as the thyroid gland. It is a mixture of the thyroid gland and contains the same amount of iodine as the thyroid gland. To avoid confusion in dosing always use the official thyroid.

Dose 0.065 to 0.12 Gm (1/2 gr) daily

**B Thyroid Thyroxine Sodium** This is an advantage of the crystalline thyroxine over desiccated thyroid. It is approximately 100 times as potent as thyroid and so small changes in dose may lead to toxic levels. Thyroxine may be administered orally but is almost absorbed by this route.

Dose 0.1 to 0.2 mg (1/200 to 1/100 gr) daily

# PARATHYROID HORMONE

Parathyroid hormone is a potent substance derived from parathyroid glands. It cannot be employed in any form other than a fluid and is rapidly destroyed by the body. It is a potent substance derived from parathyroid glands. It cannot be employed in any form other than a fluid and is rapidly destroyed by the body.

Parathyroid hormone has a major effect on calcium and phosphorus metabolism. Its effect is to increase the release of calcium from bone and to increase the release of phosphorus from bone. It also increases the release of calcium from bone and to increase the release of phosphorus from bone.

Because of its effect on calcium and phosphorus metabolism, parathyroid hormone is used in the treatment of hypoparathyroidism. It is also used in the treatment of hypoparathyroidism. It is also used in the treatment of hypoparathyroidism.

Preparations Available

**A** Parathyroid hormone (U.S.P.) and is used only in acute postoperative hypoparathyroidism (it is not for the treatment of parathyroid gland).

Preparations Available

**A** Parathyroid hormone (U.S.P.) Aqueous solution contains 100 units per 100 units. Dose 100 to 500 units (0.5 to 1.0) 3 to 5 times daily.

**B** Parathyroid hormone (A.T. 10) Each contains 125 mg of parathyroid hormone in oil solution.

Dose 1 to 3 (1/2 to 1) A.T. 10 daily orally initially then as needed with (16 mg) 1 to 2 times weekly (see pag 373)

**C** Calcitonin (U.S.P.) (V.M. 1000 D<sub>2</sub>) 1.5 mg (1/40 to 1/12 gr) 3 to 7 times per day of 10 to 20 mg (1/2 to 1/4 gr) (see pag 373).

## THE ADRENAL HORMONES

## ADRENAL CORTEX

The hormones of the adrenal cortex are all steroidal substances. To date over 30 different steroids have been isolated and identified from animal adrenal glands or adrenal venous blood. The vast majority of these steroids have no demonstrable metabolic effect. Those with metabolic effect are shown in the table below.

Metabolically Active Adrenal Steroids

Compounds isolated	Metabolic Effect	Clinical Use	Availability
Deoxycorticosterone	Na <sup>+</sup> and H <sub>2</sub> O retention increased K <sup>+</sup> excretion	Maintenance therapy Addison's disease	Readily available
Aldosterone (Electrocortin)	20 times as potent as DOCA	same as DOCA	Not available
17 hydroxycorticosterone (compound F hydrocortison)	Cabohydrate protein metabolic effects as produced by ACTH (in approximate order of their potencies) Also mild effects on salt and H <sub>2</sub> O metabolism (All have an oxygen atom at C <sub>11</sub> in steroid nucleus)	As effective as cortisone (see below)	Readily available but expensive
17 hydroxy 11 dehydrocorticosterone (compound E cortisone)		1 Useful in many diseases (see page 423) 2 Maintenance therapy Addison's disease	Readily available but expensive
11 dehydrocorticosterone (compound A)		None	Not available
Corticosterone (compound B)		None	Not available
Androstenedione 11 hydroxy androsterone 17 hydroxyprogesterone androstosterone	Androgenic (similar to testosterone but much less potent)	Not employed as an androgen	Not available (Testosterone used see p. 429)
Estroene	Estrogenic	Not employed as such	See page 421
Progesterone	Progestational	See page 422	See page 422

Questions have been raised as to whether or not all the steroids isolated from the adrenal cortex are truly naturally occurring or whether they are artifacts produced in the chemical laboratory. Recent isolation of hormones from blood obtained by catheterization of renal veins shows that most of the hormone (about 90%) is 11 hydroxycorticosterone (Compound F). In general it may be stated that the first demonstration of the effects of adrenal cortical hormone or hormones is that seen following ACTH administration (see page 423).

Clinical Preparations

Of the adrenal steroids isolated, three have had significant clinical use and trial

- A Desoxycortone Acetate U.S.P. Desoxycortone Acetate B.P. (Doc<sup>®</sup>) It only significant metabolic effect are sodium and water retention and increased urinary potassium excretion. In this respect it is approximately twenty times as potent as cortisone. It has no effect on carbohydrate or protein metabolism.
- B Cortisone (Compound E of Kendall) (17-hydroxy-11-dehydrocorticosterone)

1 The principal metabolic effects of cortisone are

- a Retention of some sodium and water
- b Increased retention of nitrogen potassium and phosphorus
- c Increased blood sugar and ability to maintain blood sugar levels during fasting in Addisonian patients
- d Return of EEG pattern to normal in Addisonian patient
- e One of the most important effects is the adrenal cortical atrophy which results with prolonged use. This is probably due to dog nose ACTH inhibition and may result in the absence of the normal response of the pituitary-adrenal axis to stress.

2 For clinical effects and uses see p 423

- C Hydrocortisone (Compound F of Kendall), 11-hydroxy-21-on-terro. This compound has recently become available for oral and local (gingival) use. Its actions are similar to those of cortisone. It is probably about  $1/2$  times as potent as cortisone on a weight basis. The metabolic effects of hydrocortisone appear to be identical with those of cortisone.

- D Whole Cortisol Extract. A water-soluble extract of the adrenal gland. Although its steroid content (if any) and mode of action are poorly understood, this agent appears to be of value in the management of adrenal crisis. Recently an attempt has been made to concentrate adrenal cortical extract in an oily solution. The resultant product, lipo-cortisol extract, is also useful, but due to the low absorption (from the stomach) and due to the fact that it may not contain some of the essential adrenal cortisol substance, it should not replace aqueous or oil extract. Lipo-cortisol extract has been shown to contain mainly compound F (about 2 mg/cc) with less compound E.

Preparations Available

- A Desoxycortone Acetate U.S.P. Desoxycortone Acetate B.P. (Doc<sup>®</sup>) or Desoxycortone Triethylacetate. Used only for supplementary maintenance of Addison's disease.
- 1 Since tablet is 2 mg tablet for absorption of water-soluble membranes. Doc<sup>®</sup> is ineffective when swallowed.
- Dose: 1 tablet daily dissolved in the buccal solution. It is almost equally effective in a given dose when injected.
- 2 Solution in a same oil 5 mg (12 g) per cc.
- Dose: 1-3 mg (10-12 g) 1 M daily for maintenance.
- 3 Hydrocortisone U.S.P. 75 mg (14 g) or 125 mg (2 g) for subcutaneous replacement.
- Dosage: One 75 mg tablet for each mg of Doc<sup>®</sup> required by the action up to 3 mg (14 g) per day. If requirements

## 416 Epinephrine

by injection exceed 3 mg (1/20 gr) one additional pellet should be implanted for a requirement of 5 mg (1/2 gr) per day by injection (implant 6 pellets) Duration of action 6-8 months

4 Desoxycorticosterone trimethylacetate 20-30 mg I M once a month

B Adrenal Cortical Extract N N R May be administered I M sub ut or I V Used in treatment of Addisonian crisis Dosage 10-100 cc (5-20 dr) or more daily as indicated

C Lipo-Adrenal Cortex Sterile Solution (C A) Administered I M only

Dosage 5 cc (1 1/4 dr) I M daily during crisis in addition to aqueous adrenal cortical extract 1-2 cc (16-32 dr) daily for maintenance

D Cortison (Compound E) (17 hydroxy 11 dehydro corticosterone acetate) See page 423

E Hydrocortisone (Compound F) (17 hydroxycortisone acetate) See page 423

## ADRENAL MEDULLA

Until recently it has been thought that the adrenal medulla secretes a single hormone epinephrine However it has been shown that extracts of adrenal medulla of cattle (U S P Reference Standard) contain two closely related hormones i.e. epinephrine (about 80%) and nor epinephrine (about 20%) The two have different actions as outlined below

Substance	Blood Vessels	Cardiac Output	Blood Pressure	Blood Sugar (Glycogenolysis)
Epinephrine	Vasodilation (overall)	Increased	Elevated?	Elevated
Nor epinephrine (levaterenol)	Vasoconstriction (overall)	No effect	Elevated	Elevated 1/2 that of epinephrine

\*Vasodilator of coronary arteries

Since epinephrine may be synthetic or derived from natural sources (usually the latter) and hence contaminated with nor epinephrine the reason for some of the apparent paradoxical physiological effects of the present preparation becomes clearer

In addition to the above epinephrine causes an immediate elevation of blood sugar by inducing glycogenolysis in liver and muscle

### Epinephrine

A Clinical Uses Epinephrine is used in a great many clinical conditions including the following

- 1 Allergic conditions Bronchial asthma urticaria angio neurotic edema and others
- 2 Control of superficial bleeding especially from mucous membranes
- 3 Used with local anesthetics to slow down absorption



and testosterone propionate. Testosterone and testosterone propionate when injected (or swallowed) are partially (about 30-50%) excreted as 17 ketosteroids in the urine. Methyltestosterone is not excreted as 17 ketosteroid. In fact its administration will result in diminished urinary 17 ketosteroids due to diminished endogenous testosterone production.

**A Clinical Use** In either sex testosterone may be indicated in any debilitating disease for its protein anabolic function. In addition there are certain uses specific to the different sexes.

- 1 **Male** As replacement therapy in failure of endogenous testosterone secretion (e.g. eunuchoidism, male climacteric, etc.). Its use in psychogenic impotence, angina pectoris, homosexuality, and benign prostatic hypertrophy is without benefit.
- 2 **Female**
  - a Gynecologic conditions: Functional uterine bleeding, endometriosis, dysmenorrhea, and premenstrual tension.
  - b Diseases of the breast: Advanced carcinoma, chronic cystic mastitis, suppression of lactation.

**B Preparations Available**

- 1 **Testosterone U.S.P. (Free)**
    - a Pellets U.S.P. 75 mg (1 1/4 gr.) implanted subcut. Dose: 4-8 pellets every 3-4 months.
    - b Microsuspension in aqueous solution for I.M. use. The dosages have not yet been determined but appear similar to testosterone propionate in oil.
    - c Ointment 5 mg/Gm (2 1/2 gr./oz.) for local testosterone effect. Average dose: 0.5-1.0 Gm. locally rubbed in over 5 minutes b.i.d.
  - 2 **Testosterone Propionate U.S.P. B.P.** In oil for I.M. injection 5, 10, 25, and 50 mg/cc. Dose varies from 10-100 mg (1/8-1 1/2 gr.) daily depending on condition being treated.
  - 3 **Testosterone Cyclopentylpropionate N.N.R. (Depo-testosterone®)** in oil for I.M. injection 50 and 100 mg/cc. This preparation has a duration of action 2 to 5 times or more that of testosterone propionate. Dosage: 100-200 mg weekly to 500 mg monthly in a single dose.
  - 4 **Methyltestosterone U.S.P. B.P.** Do not use methyltestosterone in treatment of thyrotoxicosis, acromegaly, and gigantism or liver disease.
    - a Tablets 5, 10, 25 mg for oral use.
    - b Tablets 5, 10 mg for sublingual or buccal administration. There is no advantage of buccal over oral use of the hormone.
    - c Ointment 5 mg/Gm (2 1/2 gr./1 oz.)
  - 5 **Methylandrostenediol (methandriol)** It has been claimed that this is a potent anabolic agent without the virilizing effect of testosterone. There is little evidence to support the claim. Its anabolic effect is quite poor and erratic, and the evidence for a dissociation of anabolic and virilizing effects with this hormone is lacking.
    - a Tablets 10 mg (3/8 gr.) for oral use.
    - b Microsuspension in aqueous solution for I.M. use 50 mg/cc. Dose advised: 25-100 mg (3/8-3/4 gr.) daily.
- C Choice of Preparations** In view of the great number of

preparations available. It may be difficult to decide which to use. The physician should choose those preparations which are most economical to the patient and still are effective. The use of testosterone by repeated injections should be reserved only for those very few conditions where methyl testosterone should not be used where the patient must be under very close observation (preferably in a hospital) or where the dose must be very exact (e.g., research). Even in the cases in which methyl testosterone cannot be used, pellets of free testosterone may be given to the patient or relatives may be instructed in the administration of I.M. medication. Therefore, the alternatives of cholesterol, methyl testosterone, orally implantation of testosterone pellets, or intravaginally cyclopentylpropionate

## FEMALE SEX HORMONES

The female ovaries secrete two steroids with marked physiological effects, namely, estrogen (o-est- diol) and progesterone.

### Estrogen

**A Effect of Estrogens in the Human.** The physiological effects of

- 1 Proliferation of endometrium
- 2 Change in vaginal cells (cornification and lowering of vaginal pH below 4.0)
- 3 Ductal proliferation of breasts
- 4 Stimulation of osteoblastic activity
- 5 Slight protein anabolic effect
- 6 Moderate sodium and water retaining effect

**B Clinical Use.** Estrogens are useful in both sexes for their effect on osteoblasts in the stimulation of osteoporosis.

1 Female. Estrogen is used as replacement therapy in cases of oophorectomy (e.g., menopausal).

2 Male. Used as adjunct therapy of carcinoma of prostate.

**C Preparation.** Available. There are many substances that have estrogenic activity including nonsteroidal but (e.g., diethylstilbestrol, diethylstilbestrol). However, of all the steroids only certain ones are used clinically. There is no evidence that any of the estrogens are less toxic than any other. Toxicity (e.g., nausea and vomiting) is usually due to overdosage. Most of these estrogens are exceedingly powerful drugs having profound physiological effects; very small doses and also having the appropriate and toxic levels that are quite similar. The physician should select the good preparations and watch their harmful effects rather than change to new ones. There is little or no need at present to administer these estrogens by any but the oral route. Absorption seems to be very complete and there is no evidence that nausea or vomiting can be decreased by parenteral administration. There is likewise no evidence that the "naturally occurring" estrogens are any more effective than the synthetic ones. Although estrogens apparently play a role in mammary tumor of animals, there is no evidence that they are carcinogenic in human.



available little can be said as to duration of therapy. It would appear at present that prolonged administration will be necessary in many cases. Where knowledge is available regarding recommendations for treatment these are indicated in the text.

### Dangers

These agents are potentially very dangerous. However with proper caution most of these dangers can be overcome. The principle dangers are that these drugs may induce

- 1 Hyperglycemia and glucosuria (diabetogenic effect). This is of major significance in the early or potential diabetic.
- 2 Marked retention of sodium and water with subsequent increased blood volume and hypertension.
- 3 Negative nitrogen balance with loss of body protein.
- 4 Potassium loss with development of a hypokalemic alkalosis.
- 5 Hirsutism and acne (especially disagreeable in females).
- 6 Cushing's syndrome (may develop with long continued administration).
- 7 Activation or production of peptic ulcer.
- 8 Lowering of resistance to infectious agents.

### Controls to Be Employed to Correct or Minimize Dangers

- A Always reduce the dosage as soon as consistent with the clinical response.
- B It is desirable that the patient be hospitalized during the initial period of treatment of 1-2 weeks but this is not necessary in all cases.
- C During the first 2 weeks of therapy the following should be carefully observed:
  - 1 Blood pressure daily or every other day.
  - 2 Weight daily or every other day.
  - 3 Daily eosinophil count for a few days to judge initial response to medication with ACTH.
  - 4 Initial complete blood count repeat as indicated.
  - 5 Initial sedimentation rate repeat as indicated.
  - 6 Frequent urinary sugar fasting blood sugar if reducing substances are found in the urine.
  - 7 Serum potassium should be checked occasionally if large doses of hormone are to be given over more than several days.
- D All patients should be on high protein diets (100+ Gm. proteins).
- E If edema develops place patient on low sodium diet (200-400 mg. sodium daily). Mercurial diuretics may be employed when strict sodium restriction is impossible.
- F Potassium chloride 3-15 Gm. daily in divided doses should be administered.
- G In cases of long continued administration testosterone preparations (see page 420) in doses of 10-25 mg. daily may be used to counteract the negative protein and potassium balance.
- H Do not stop either drug abruptly. There may be a severe rebound of the disease process. Also remember that cortisone (or hydrocortisone) causes atrophy of the adrenal cortex probably through endogenous ACTH inhibition. Sudden withdrawal may lead to symptoms of Addison's disease.

Contraindications and Special Precautions

- A. In Patients Requiring Maintenance Cortisone (or hydrocortisone only)** Patients receiving cortisone or hydrocortisone only especially the oral preparation must be carefully watched and managed. The aim of the suppression of endogenous ACTH and subsequent adrenal atrophy then patient is unable to respond normally to stressful situations (e.g., surgery, infections, etc.). Whenever such a situation occurs or is to occur, the dosage of cortisone or hydrocortisone should be raised and/or methylprednisolone and ACTH given. If only cortisone or hydrocortisone only can be administered, it must be administered in larger doses till the crisis has passed.
- B. Hypertension** These drugs should be used with extreme caution in individuals with damaged myocardium. The increase in extracellular fluid may lead to cardiac decompensation. Always begin with small doses and with patient on low sodium diet.
- C. Liver Dysfunction** The drug is probably contraindicated should be used with extreme caution in patients with serious liver damage associated with edema and/or oliguria.
- D. Hypersensitivity to Drug** Certain diseases appear to make the individual more sensitive to these agents. This is especially true of the rheumatic diseases especially lupus erythematosus. It usually begins with low dosage and increases cautiously in these individuals.
- E. Disposition to Psychosis** The drugs cause use of small but significant amount in most individuals relieving them but some individuals (those predisposed to psychosis) may develop an atypical psychotic episode while the drug is administered. If the psychosis it should be stopped or the dose should be lowered and the patient should be fully observed and protected. Physicians have committed suicide under the influence of the drugs.
- F. Effect on Thyroid** When given for prolonged periods the drug may depress thyroid function. This may inhibit the action of ACTH on the adrenal cortex and possibly the action of cortisone on the metabolism of the body. Give supplement of thyroid 85-200 mg (1-3 gr) if the drug are to be given for more than 3-4 weeks.
- G. Effect on Peptic Ulcer**
1. **Asymptomatic** is a contraindication to use of the drugs because of danger of perforation or hemorrhage.
  2. **Old peptic ulcer** The agents stimulate the ulcer. They should be used in the presence of this disease only as an emergency to avert it and with optimum anti-ulcer therapy.
- H. Tuberculosis** Active or recently healed tuberculosis is a contraindication to the use of the drug.
- I. Infection** Because the drugs tend to lower resistance and therefore promote dissemination of infections they must be used with extreme caution even when appropriate antibiotics are being given, in a pyogenic or chronic infection.

- E. Surgical Exploration** (e.g. simple incision, thoracotomy or laparotomy). It may be indicated as a final evaluation measure. In many cases the surgeon must be prepared to perform a radical surgical operation if macroscopic or frozen section examinations indicate malignant disease.
- F. II examination.** Upon completion of the clinical studies, *emphatic reassurance of the patient regarding the negative findings is necessary.* If findings are equivocal, the patient should be kept under close follow up observation with appropriate diagnostic measures.

### Suggestions for Treatment

#### A. Factors Influencing Choice of Treatment

1. Nature (inherent characteristics) of the given neoplasm: rate of growth, cytological characteristics, invasiveness, amenability (e.g. radiosensitivity or radiocurability), tendency to metastasize, and nature of metastasis.
2. Age of patient.
3. Physical and emotional status of patient.
4. Patient's ability and/or willingness to cooperate with the prescribed therapy.
5. Availability of professional and technical facilities.
6. Stage of the tumor at the time the patient is first seen.
7. Location of the lesion. Proximity to vital or tubular structures.
8. Secondary complications of the disease. Local pressure symptoms, hemorrhage, systemic effects of the primary lesion and the metastases.
9. Functional, cosmetic, and psychological effects of therapy.
10. Patient's ability to tolerate radiation therapy (i.e. tolerance of solar or other radiation).
11. Cost of therapy.

#### B. Treatment of Benign Lesions. The physician's clinical impression of the benign character of lesions must always be verified by biopsy and microscopic examination.

1. Simple eradication of the tumor by surgical technique (including curettage and cauterization) is usually the preferred method of treatment. Radiation technique may occasionally be employed.
2. General indications for removal of benign tumors:
  - a. Diagnostic purposes (possibility of malignancy)
  - b. Pressure on vital structures
  - c. Obstructive symptoms
  - d. Mechanical (static) deformities
  - e. Pain or other marked discomfort
  - f. Systemic effects (e.g. hormonal)
  - g. Hemorrhage (acute or chronic)
  - h. Cosmetic purposes
  - i. Psychological purposes (reassurance)
3. More extensive surgery. The surgeon must be prepared to perform radical surgery if macroscopic appearance or frozen section examination indicate malignant disease.

#### C. Treatment of Malignant Lesions

1. Primary lesion
  - a. Complete eradication of the primary lesion by surgical

(includi g urettag and c t rization) or radi tion  
m thods mu t be att mpted wh ever pos sible

- b Radical su g ry Clinical evidenc of r gion l m taste  
sis may ind t need for r di ls r gi al removal of th  
primary tumor and th l l d node  
S r gi al r move) of the primary t mo m y attil be ind  
m t d when m taste ses re sy temic b ts e gr wing v ry  
lowly ( g thyroid carcinoma)

d R dition ther p y m y be us d to arr st or slow the  
p og ss of th d t e if the tumor is r di se sili e

e Ch mothe apy Se below

## 2 Metastatic lesions

S gicate ci ion may be of val e wh le ions s il  
tary slowly growing p infu, or when they produc other  
ute symptoms (obstru tion ic )

- b Radi tion ther py is indi t d if l ions re radios sensitive  
a d pa ticu ly if they a m ltipl dise min t d  
Chemoth rapeutic m thods may be employed u ing the  
spe ific ag t which r known t aff ct c t l n types of  
p im y and m taste t mor The ag ni ar ordi  
n rily withheld v) the ts d lntlu a d r sympto  
matic rell f

(1) Androg ni nd st ogenic t roids Definit bene  
fici l f t c have been ob rved with the m p l c l  
u of the st r lds in certain neopla ti di eas e  
but m ch of th work r m in on an pe im t l  
b is Th d tion of eff cts unknown "steroid  
thera y fa n uen r rative and shos d aser  
replace radical surgery e oremable carcinoma

(1) E trog Th d ge of the e t oge sm l be  
indiv d d ording to the pati s espon  
and the t l ty of th dr g (shor t m u a  
di rbe d maitia nd d ms)

(1) Soft tissue m t st from bre st a loma  
(t lung brain et ) T anst t improve  
m m ocure i mall per t ge of ld rly  
pati nia In gen r l, estrog re r served  
for pat ts b or mor y rap at the meno  
p u e Give Di thylate bestrol t S P d 30  
mg f usually 10 15 mg l or Enuyl E t adi l  
N y R d 2 0 3 mg orally dily Cyclic l  
admi t tim f i 40 days on 10 d y off)  
is commended

(2) F cat tic r i ome nd m taste a (see p ge  
109)

(b) And og a M thylt test rone t S P l 10 mg  
sub i gually daily or T test rone P opnat  
U S P Ts 200 mg l M 3 times w lly p r n  
may be indi t s for

(1) Car inoma of th t or ov y Give  
partial or comple r li f of p m f r vari bl  
pe lsd but no objective lmr vesent

(2) R m a chosen qd let l m t case in 15%  
of e se how lmp ovement but on y occasional  
improven st is the ved in soft tissue met sta

- (2) Nitrogen mustards Although employed with benefit in certain cases of metastatic carcinoma these agents have proved most beneficial in certain diseases of the blood and lymphatic systems (see page 241)
  - (3) Mustard like compounds (TEM TEPA) Similar to the above although less toxic (see page 238)
  - (4) Antimetabolites (aminopterin 8 MP) See pages 231-239
  - (5) Urethane® See page 239
  - (6) Radioactive salts Effects are due to radiation rather than chemical action
3. When none of the above procedures is possible
- a. Narcotic drugs Liberal but judicious use especially in advanced and terminal malignant disease
  - b. Surgical measures
    - (1) Relief of specific symptoms Surgical intervention (e.g. tracheotomy thoracentesis paracentesis lumbar puncture etc.) may be necessary to control progressive or emergency obstructive or other pressure symptoms
    - (2) Nonspecific surgical methods (hormonal modification)
      - (a) Adrenalectomy Bilateral removal of the adrenal glands can sometimes produce a substantial regression of metastatic and widespread male and female mammary cancer. Although there is objective as well as subjective evidence of improvement the relief is most often of temporary nature. This is still largely a research technique since it is a major operative procedure expensive and requires careful follow up steroid replacement therapy. The use of this procedure in the treatment of other neoplasms is being investigated but no significant statistics are available at present.
      - (b) Ovariectomy Removal of the ovaries has been advocated for some time as a treatment for advanced breast cancer. The results of the operation are usually transient and the relative efficacy of the treatment has been questioned.
      - (c) Orchiectomy Castration may result in significant regression of primary and secondary tumors of the prostate and male breast. In patients who fail to respond to orchiectomy subsequent adrenalectomy may prove to be effective. Subjective and objective relief may persist for more than a year.

#### D. General Problems

##### 1. Explanation to the patient

- a. Factors of importance Opinion varies greatly as to whether or not it is advisable to inform patients that they have malignant neoplastic disease. This matter must be individualized and must naturally vary with the temperament intelligence attitudes and desires of the patient. Under certain circumstances it may be necessary or advisable to inform the patient as to the true nature of his condition irrespective of the above factors.
- (1) If the patient demands an explanation of his illness

- (2) If the patient's economic status requires a forewarning  
 III permit proper disposition of estate, etc.
- (3) If the patient refuses to carry through on a prescribed  
 diagnostic and/or therapeutic regimen.
- (4) If the neoplasm is growing relatively slowly, is non-  
 invasive and fairly treatable.
- (5) If the patient expects (or threatens to shut) his  
 finances in a sea for a cure.

III. Manner of explanation. If explanation is indicated, use  
 mild term such as growth, lump, or even  
 tumor, but in most cases it is advisable to avoid the  
 term cancer. Be guarded in statements as to prog-  
 nosis and lean towards the optimistic side. *Always*  
*offer some ray of hope.* When the clinical situation  
 is not utterly hopeless, a cheerful optimistic and r-  
 assuring attitude may do much to allay the fears and ap-  
 prehension of the patient and the family.

2. Explanation to the family. It is often advisable to inform a  
 near relative (preferably the wife, when this is feasible) of  
 the nature of the illness and the prognosis. The qualifying  
 facts mentioned above should be kept in mind in deciding  
 who, how, and what to tell.

3. Provision for chronic and terminal care.

- a. Assistance of social agencies. In view of the chronicity  
 and the psychological and socio-economic implications of  
 the illness, the help of a medical or social agency is  
 advisable in appropriate cases.
- b. Hospital or nursing home care may be indicated.  
 Home care. If patient and family decide on home care,  
 it will be necessary to instruct one or more members of  
 the family in the technique of administration of drugs  
 (especially parenteral injections).

## TREATMENT OF ADVANCED MAMMARY CANCER

	P m ope l	Posim op l
<b>EXCISION OF GOVADS</b> <b>(OVARIECTOMY)</b> ↓ Elimin t i fun tion	Imp m nt i f w Be ficial N ly i at l ger than 6 12 m the This m thod id m d at th p t tim	E l eff cti e than to prem op sal an r
<b>OVARIAN IRRADIATION</b> ↓ Elimin t e rian fun tion	B f i l a h m f t mbe of f pe lod up to m y o m r Be t e its i bo y m t t e (69%) and p lm ry and pl u al l ion (50%)	Le if t than to p m nup }
<b>ANDROGENS</b> ↓ P ot in an b li ff t (†) Ov ian pp i n	Symptom is h f in 65% of p t t and obj ti imp vem t i oe ous and soft ti m t i in 20% of pati t p rh p ight p along tion of lif e w ll p hli tive ff t	L uffi t t xpe
<b>ESTROGENS</b> ↓ Empi i l ff t	V i bl po E t e hould p obably be d OVL Y s m y r ff m op e	S by ti imp m t in bout 60% of obj tive improve t in 25% of cases E t i te f v o ably ponding t t m m y h om p elongation of lif
<b>ADRENALECTOMY (BILATERAL)</b> ↓ El mi t p odu tion f and e lik and at g lik t t de (P t t m int in d op rti en and D O C A aft op tion )	In uffi t sp i	S by ci ve obj ti imp m t in l m t d un be of p ti te t t d with thl th spy i at





## Chapter 17

# VENEREAL DISEASES

## SYPHILIS (Lues)

An acute or chronic disease caused by infection with *Treponema pallidum*. It may be either congenital or acquired. The acquired form of the disease is usually transmitted genitally but may be acquired by extragenital routes.

### DIAGNOSTIC FEATURES

#### Primary Syphilis (code No. 147)

- A History of contact with an infected individual 1-6 weeks (usually 3-4 weeks) prior to appearance of primary lesion.
- B Primary lesions are pleomorphic, may be single or multiple and are usually located on the external genitalia, although extragenital chancres are not rare.
- C Three or more carefully performed dark field examinations (on successive days) are necessary before a final report of negative may be made.
- D Both complement fixation (e.g., Kolmer) and precipitin (e.g., Kahn) tests should be performed. Quantitative blood tests (performed by a reliable laboratory) since they may demonstrate changing titers are preferred for both diagnostic and follow up purposes.
- E Regional lymph nodes on one or both sides are often rubbery, discrete and non-tender.

#### Secondary Syphilis (code No. 013-147)

- A Usually occurs 7-10 weeks after exposure to the disease and 2-3 weeks after appearance of the primary lesion.
- B There is often evidence of systemic involvement with fever, generalized lymphadenitis, non-pruritic maculopapular dermatitis, nasopharyngitis, laryngitis, conjunctivitis, alopecia, arthralgia, mucous patches and condylomata.
- C Blood tests for syphilis are almost invariably strongly positive.
- D Cutaneous and mucous membrane lesions may show *Treponema pallidum* on dark field examination.
- E Spinal fluid usually shows transient involvement.

#### Relapsing Syphilis

- A Usually occurs within 6 months to 2 years after onset of the disease.

- B Often follows inadequate or improper therapy (e.g. penicillin for coexistent gonorrhea)
- C Blood tests for syphilis usually revert to a positive reaction or if already positive to an increasing serologic titer (based upon quantitative blood tests)
- D Relapse may be of a variety of clinical types. The commonest of these are mucocutaneous, CNS, ocular, and neurological (the latter in the absence of clinical evidence)

Latent Syphilis (code No. 900 147) (Exclusively less than 4 years latent, more than 4 years)

An intermediate or quiescent phase after secondary lesions have disappeared and while tertiary symptoms are not yet evident

- A Latent syphilis offers no clinical evidence of disease other than the positive blood test. It is therefore important to rule out the positive blood tests the most common causes of which are technical or clerical errors, acute fever, yaws, infectious mononucleosis, malaria, leprosy, leishmaniasis, smallpox, vaccinations, and lymphogranuloma venereum

Never make a diagnosis of latent syphilis solely on the basis of a single blood test. Rule out the possibility of the above factors. If the blood test is only very transiently and weakly positive, the diagnosis of lues should be questioned. Conversely, if the blood test is persistently positive for 3 or more months, lues is the most likely diagnosis.

- B Spinal fluid must be completely negative

Late (Tertiary) Syphilis (code No. 814 147)

Involvement may be diffuse, may be confined to certain organs, system, or may be localized as discrete granulomatous lesions (gummas) in any and all tissues

- A Macrocystic Gummatous lesions of the skin and mucous membranes
- B Cerebral Diffuse or gummatous lesions of bones and joints with periosteal arthritis, synovitis and osteomyelitis
- C Ocular Conjunctivitis, iritis, uveitis, choroiditis, keratitis and retinitis
- D Visceral (including diaphragm) Gummatous or diffuse involve ment of liver, pancreas, lungs, spleen, kidneys and stomach
- E Cardiac
  - 1 Compensated aortic stenosis (code No. 481 147)
  - 2 Aortic regurgitation (code No. 433 147)
  - 3 Aneurysm (code No. 481 147 6)
- F Neurosyphilis

- 1 A syndrome is neurosyphilis characterized by spinal fluid abnormalities (Group I and II see page 438) but without evidence of symptoms or signs of neurological involvement
- a May be classified according to CSF changes (see page 43) as mild, moderate or severe (Groups I, II or III)
- b However, it may develop later in the neurosyphilis cycle or during an apyretic phase of lues
- 2 Symptomatic

Acute syphilitic meningitis (code No. 812 14 6)

(1) Less likely to occur within 2 years after infection

Special Considerations Regarding Penicillin Therapy

- A Herxheimer's reactions usually of a mild degree occur with marked frequency and consist of fever and generalized aches and pains within 24 hours after onset of therapy
- B Some clinicians feel that in late syphilis it is necessary to administer a course of bismuth and iodides prior to penicillin therapy in order to diminish the hazards of Herxheimer's reaction or "therapeutic paradox." These dangers, if they exist at all, are minimal.
- C Sensitivity to penicillin (see page 305) contraindicates further use of the drug.
- D Relapse following one or more courses of penicillin therapy requires consideration of other therapeutic agents.

Aureomycin® Treatment Methods

Oral Aureomycin® has been reported to be effective in the treatment of syphilis, but clinical experience with the drug is not extensive. Optimal dosage schedules, toxicity, failure rates, etc., remain to be determined. One Gm. every 4 hours day and night for a total of 70 Gm. has been suggested. Its use may be considered in those patients sensitive to penicillin.

Bismuth Treatment Methods

- A Indication
  - 1 As adjunct or supplement to penicillin therapy enhances antiluetic activity of that agent.
  - 2 Preliminary to penicillin therapy in latent or late lues when Herxheimer's reaction is feared.
  - 3 In cases of multiple antibiotic sensitivity.
- B Contraindications, Severe stomatitis or severe renal damage.
- C Drugs Available
  - 1 Bismuth Subsalicylate Injection, U.S.P. 0.2 Gm. 1 Ml. once weekly.
  - 2 Iodobismuthite sodium with ethyl aminobenzoate (Iodobismitol with benzocaine) 2 cc. 1 Ml. every 3 days. 20 injections comprise a course of treatment. If necessary, such therapy may be continued over a long period of time (2 cc. 0.025 Gm. metallic Bi).
- D Technic of Intramuscular Injection with Bismuth
  - 1 Cleanse skin of outer upper quadrant of buttock with alcohol sponge.
  - 2 Agitate bismuth vial vigorously for one minute to ensure a uniform suspension.
  - 3 Aspirate bismuth suspension in a 2 cc. syringe through a 1 inch No. 20 gauge needle.
  - 4 Replace the 1 inch needle with a 1½ to 2 inch No. 20-22 gauge needle.
  - 5 Draw the buttock downward with the free hand and with a wrist motion only plunge the needle quickly into the muscle. Insert needle more deeply if necessary.
  - 6 Aspirate with the syringe for 10 seconds to make certain that no blood vessel has been entered. If any blood appears, choose another injection site.
  - 7 Inject bismuth suspension slowly and steadily.
  - 8 Detach syringe from needle and inject ½ cc. of air.

- 8 Withdr w ne dl with a single rapid mov ment  
10 Massage ( r have patient massag ) the ar a firmly fo about  
5 minute

# DIFFERENTIAL DIAGNOSIS OF VENEREAL DISEASES

Dis ase	Organism and How Demon t d	T st	Lesions	
			Re ho	Ge t
Syphil	T pon map p lld m (Dark fi ld exam)	C mplem t fixa tion ( g Kolmer) and p ipitin ( g Kahn) t t	h n fluctuant	Painl ulcers
Chan oid Hemophilus ducreyi (G am at i )		None	Us ally fluctuant	P ainul ulce s
Lymphog anuloa erereum virus (Cult e method )		Compl m t fixation t t	Usually fl ctuant	P inl s evanescent ulcer
G an l ma inguinal Dono an bodi ? (W ight talo)		Non	U ually none	Painl spreadi g ul r
Gonorrhea (G am stain)		Compl m nt / za tion ( alu ?)	U ne	U th ti
N is la gonor here				

## GOVORRBEA

Con rhea is an a ute or hronic infe ti us d se ca ed by th gram negati diplococcus Neisseria gonor hea s d p acic lly alway t anemitted among adults by a ual into cou se Acut gon rhea in the ad it male is cha ct ri ed by an acute ur thrills with painful urination and p ul nt u ethral discharge Chronic gon rhe may be manif et d by chronic inflammation of the th a proat e pididymis and semin vesicl s but ra lly if eve of the upper u thary t a t Conor hea in the f male begins in the urethra, vagine and v ginal glands and is ha acterited by painl i m ination and purul nt dis harg Commonly th infe tion prevails to the vi rus tube and othe pelvic tract res cau ing abdominal pain and tenderness with evid of con titutional symp tom Syst mic inf ction with a plicermia and manif eted by ndo ard ti o arthriti is ) s common The o gani m has a at ong affinity for the ocular m ous m embran s and may caus a s ious and b lading ophthalmia

### Di gnosis

A llw ry of genital dis ha ge and dy u is occurring d to 10 days following a ual int course with an inf cted ind vidual Symp tom will vary w th the anatomic structures involved Demon str ation of the gr m negative int acell lar diplococcus in ru dal from e ions by st in d (G am o methyle e blue) sm and by cultur Blood cultur s m y be positive in p tient with septi mia Conpl mnt fixation t t may be pos tive s ve al w la aft r the in ti i inf ction  
M Gon t i T ct level some t

- 1 Urethritis acute anterior (code No 744 103)
  - a Smear and if necessary culture of material obtained from the urethral meatus will demonstrate the causative organism. This is obtained by urethral stripping and never by any other method during the acute phase. The discharge is universal in the chronic phase.
  - b Two glass test. Acute first glass is cloudy the second is clear. Chronic both glasses contain shreds.
  - c Clinical symptoms are most often acute and definite. Prostate and seminal vesicles may become involved.
- 2 Prostatitis acute (code No 754 103). Urethral discharge may or may not be increased. Smear and culture findings will be as above. Perineal pain is common and is increased by defecation. Low back pain may be present. Constitutional symptoms such as fever and chills may be present. Dysuria is frequent and retention may occur.
- 3 Acute epididymitis (code No 756 103). History of current urethritis. Smear and culture findings as above. There is also testicular pain swelling warmth and redness.

#### C Female Genital Tract Involvement

- 1 Acute urethritis (code No 740 103). Smear and (preferably) culture of urethral and vaginal discharges should be performed. There is redness and swelling of vaginal vestibule and external meatus.
- 2 Chronic gonorrheal infections (after 4-6 weeks)
  - a Very careful and repeated cultures and stained smears of material obtained from Skene's Bartholin's or endocervical glands. (For technique see U.S.P.H.S. VD bulletin 97 1945.)
  - b Pelvic inflammation (code No 066 103) may be characterized by lower quadrant abdominal tenderness mass peritoneal irritation and systemic manifestations.

#### Treatment

Penicillin streptomycin Aureomycin® Terramycin® and the sulfonamides are all effective anti gonococcal agents although penicillin is generally the drug of choice.

#### A Acute or Chronic Uncomplicated Urethritis (male or female) (code No 740 103). Avoid all local treatment such as irrigations manipulations and instillations.

- 1 Penicillin therapy. Several effective techniques are available. Always draw preliminary blood specimen for serological test for syphilis and examine patient clinically for evidence of syphilis since the danger of masking early syphilis by penicillin treatment is very real.
  - a Crystalline Penicillin Procaine in Oil Injection U.S.P. 600 000 units I.M. as a single injection a convenient method for ambulatory patients.
- or b Penicillin (aqueous) 30 000 60 000 units every 2 hours for 5 doses (total of 300 000 units).
- 2 Alternative therapy. If coincident lues is suspected the above treatment should be altered as follows
  - a Penicillin. Reduce total dosage of penicillin to no more than 150 000 units.

or b Sulfonamid treatment.

(1) Sulfadiazine or sulfathiazole 4.0 Gm (60 gr) orally Stat followed by 1.0 Gm (15 gr) every 4 hours day and night for 5 days for hospital patients

(2) Sulfadiazine or sulfathiazole 1.0 Gm (15 gr) orally 4 times a day for 5 days for ambulatory patients (30-40% failures by this method)

or c Combined penicillin (reduced dosage) and sulfonamid

3 Follow up. Should consist of examination of the patient at weekly intervals for at least 3 weeks or preferably

a Weekly examination for evidence of urethral discharge, pain, or rash

b Stained smear and if possible culture of any inflammatory exudate weekly. Avoid prostatic massage, urethral swabs or instrumentation as a means of obtaining material for examination in acute cases

Blood test for syphilis and examination for clinical evidence of late at the end of the third week and again at 3, 6, 12, and 24 months

4 Retreatment of penicillin failures (suspect other etiology)

If any of the weekly checks shows bacteriologic evidence of persistent gonorrheal infection repeat the penicillin treatment as above. Consider the possibility of urological complications. If such can be reasonably excluded consider treatment with

a Increased dosage of penicillin

b Combined penicillin and Monomid utilizing a combination of the procedures mentioned above

c Streptomycin 0.3-0.5 Gm I.M. as single dose

d Aromycin® 1.0 Gm orally Stat and then 0.5 Gm at 6 hours later also for 4-6 doses

Tetracycline® 1.0 Gm orally Stat and 1.0 Gm repeated in 6 hours

5 Miscellaneous failures. Often associated with treatment of the

sexually related diseases this is because of therapeutic failure and

the public still generally has a tendency to believe that modern treatment

is infallible. It is now recommended that case be treated in nature

The danger of this concept is the misapplication to the patient

B Acute Gonorrhea Penicillin (Code No. 764-103) Treatment

1. Not all cases and the utilization of the urine may produce

2. Symptomatic relief

C Acute Epididymitis (Code No. 738-103) Above treatment in

1. acute

2. Redness

3. Cold compresses to the scrotal region

4. Analgesic for relief of pain

5. Supportive to be used during convalescent and last 24 hours

D Prostatic Inflammation (Code No. 738-103) (Acute Gonococcal Prostatitis)

1. Acute

2. Absolute rest

3. If necessary, analgesic during acute phase

- Examine carefully for clinical evidence of lues. Draw blood for serological test.
- d Penicillin 50 000 100 000 units every 3 hours day and night for a minimum period of 5 days or until patient is afebrile for 2 days
  - (1) Convalescent period. If patient becomes afebrile and asymptomatic keep her at bed rest until WBC and sedimentation rate become normal (may take a month or more). Observe the patient during and following her next menstrual period for pain and pelvic changes. If these are absent discharge her in home care on the convalescent program outlined below.
  - (2) Retreatment. If symptoms fever leukocytosis increased sedimentation rate or positive vaginal smear persist or if they recur at the time of menses administer a second course of penicillin.
- e Retreatment with sulfonamides. If the patient fails to respond to 2 courses of penicillin therapy give 4.0 Gm (80 gr.) of sulfadiazine or sulfadiazine-sulfamerazine mixture and follow with 1.0 Gm (15 gr.) every 4 hours for 5-8 days. Give equal or double amounts of sodium bicarbonate with the sulfonamides. Observe usual precautions for sulfonamides.
- f Pyrotherapy. In the rare case that fails to respond to penicillin and the sulfonamides pyrotherapy may be of value. This must be given only by experienced personnel.
- g Convalescent program. After the patient is discharged from the hospital give the following instructions:
  - (1) Sedentary life.
  - (2) No sexual intercourse until signs and symptoms have completely cleared (usually takes about 6-8 weeks).
  - (3) Douches. Prolonged douches of warm tap water using 1-2 gallons and administering slowly and gently over a 15-20 minute period once or twice daily. The patient can perform this procedure most effectively while sitting in the bathtub.
- 2 Subacute (or acute exacerbation of chronic form)
  - a Absolute bed rest until signs and symptoms have cleared.
  - b Douches as above.
  - c Penicillin is much less effective in this phase of the disease but a trial of therapy is warranted (see above).
- 3 Chronic (chronic gonococcal salpingitis code No 787.103.0)
  - a Bed rest during acute exacerbations.
  - b Penicillin is usually ineffective.
  - c A course of pelvic diathermy treatments may be of value.
  - d Surgical procedures may be indicated. This decision should be made by a gynecologist. Results of surgery are not uniformly satisfactory.

#### GRANULOMA INGUINALE (code No 148.199)

A chronic contagious disease caused by infection with an organism of as yet unproved identity and known as the Donovan body. It is manifested by a painless sharply defined reddish verrucose

and ulcerate easily bleeding, and is anatomous to the lesion of the skin or mucous membrane of the genital region. If untreated the lesion gradually extends to involve the entire genital area and later the ed, ce t are s of the abdomen and thighs. It rarely involves deeper structures. There is usually no lymphadenopathy. Lesions rarely heal spontaneously although the process may remain stationary for years. Deep tissue scrapings or punch biopsy of peripheral granulation tissue should be stained with Wright stain and examined for Donovan bodies (see Table on page 443).

#### Treatment

1. Aureomycin® and chloramphenicol are both effective. 1 Gm daily for 1-2 weeks may be tried.
2. Streptomycin is highly effective but may be contraindicated by the danger of damage to the vestibular apparatus. If used, it may be used in dose of 1 Gm IM daily until the lesion is healed.

### LYMPHOGRANULOMA VENEREUM (code No 53 198) (Lymphogranuloma inguinale or Lymphopethis Venereum)

Lymphogranuloma venereum is an acute or chronic venereal disease caused by a specific virus. It is characterized by minimal herpetiform genital lesions and may be complicated by regional lymph node involvement and at times by variable constitutional reaction.

The incubation period is unknown (days to months). Initial lesions are small or unnoticed, herpetiform or ulcerative and may appear on any part of the external genital area. Inguinal buboes appear 1-4 weeks after infection, are often bilateral and may or may not be suppurative. The nodes may fuse, soften and break down, forming multiple sinus tracts. Extensive scarring may occur. Chronic ulcerative disease manifested by rectal pain, sanguine purulent discharge and rectal strictures is more frequently encountered in males than in the patients. Constitutional reactions frequently accompany the stage of bubo formation and are characterized by fever, malaise, anorexia, malaise and neurological manifestations. Consider this disease as a possibility in undiagnosed cases.

The Frei test (in 2 embryos are genital) is of value. If one previous to the start of 4 weeks duration a negative reaction probably rules out lymphogranuloma venereum. A positive skin reaction may mean an active infection, past (old infection), or related viral infection (false positive). There may be a reversal of the albumin reaction due to the serum. Complement fixation tests are of doubtful value. A skin test is the possibility of primary infection (see page 439). If positive (usually weakly positive) a cure may or may not occur.

#### Treatment

##### 1. Systemic Therapy

1. Aureomycin or sulfathiazole 1 Gm. (15 gr) 4 times for 1-2 weeks or longer probably has no effect against the virus but is effective in preventing secondary complications.



## 448 Chancroid

- 2 Aureomycin® 0.25 to 0.5 Gm (3 3/4 to 15 gr) orally q i d for 5 to 14 days has been reported to be beneficial

### B Local Measures

- 1 Bed rest (provides local comfort)
- 2 Warm fomentations to buboes p r n for discomfort
- 3 Analgesics p r n
- 4 Aspirate fluctuant nodes under aseptic precautions (see below). Incision and drainage are to be avoided (to prevent lymphatic obstructions)
- 5 Proctoscopic examination for diagnosis and for later evaluation of changes
- 6 Extensive plastic surgical repair operations may be necessary in the chronic and rectal form of the disease. Rectal strictures should be treated by prolonged gentle dilation although in extreme cases this may be impossible and colon shunting procedures may be necessary

### CHANCROID (Soft Chancre)

(Of Penis code No 751 10x) (Of Vulva code No 774 10x)

A venereal disease caused by *Hemophilus ducreyi* and manifested by painful genital ulcer or ulcers often complicated by suppurating inguinal lymph nodes (buboes). Incubation period is from 3 to 5 days (range 2 to 7 days?) following venereal exposure. The genital lesion begins as a macule or vesicopustule which ruptures to produce a shallow necrotic undermined ulcer. Single or multiple painful lesions may occur and phimosis may result. Regional lymph nodes become enlarged in a few days to 2 weeks and are usually unilateral, soft, fluctuant and tender. The nodes may rupture or may subside spontaneously. Giemsa stained smears from the lesion reveal *Hemophilus ducreyi* which may also be cultured from pus from the lesions of the buboes. Syphilis must be excluded by the diagnostic measures outlined under the diagnosis of syphilis (primary). The two diseases may coexist.

### Treatment

#### A Specific Therapy

- 1 Sulfadiazine 1.0 Gm (15 gr) q i d for 1 week. Observe usual precautions with the use of sulfonamides (see page 501)
- or 2 Aureomycin® or Terramycin® 0.5 Gm every 6 hours for 5 to 7 days

#### B Local Therapy

- 1 Careful cleansing of ulcerations with soap and water b i d (after diagnosis has been made) will suffice. When lesions fail to heal promptly soaks or compresses of 1:10,000 potassium permanganate solution may be necessary
- 2 Fluctuant buboes may be aspirated with a large gauge (No 16) needle as indicated. Warm compresses or a hot water bottle may be applied to the groin for comfort and to hasten fluctuation or regression of buboes



## 450 Varicella

### Prophylaxis

Active prophylaxis is not practical but passive protection or modification may be accomplished

- A Complete temporary protection of exposed susceptibles usually follows administration before the sixth day of incubation or 20 cc. of convalescent serum 2 to 10 cc. of immune serum globulin (gamma globulin) or 3 to 10 cc. of human immune globulin (placental immune) I M
- B Modification of the disease followed by permanent immunity usually results from the injection of half the above doses on the fifth or seventh days equal doses on the eighth day or double doses on the ninth or tenth days following exposure

## RUBELLA (German Measles) (code No 010 165)

Rubella is an acute communicable disease of viral origin characterized by rash and lymphadenopathy

### Diagnosis

The incubation period is 2 to 3 weeks. A short prodromal period of malaise or aching in the posterior cervical nodes may precede the fine papular eruption which appears usually first on the face and quickly spreads to the trunk and extremities. Suboccipital and posterior cervical adenitis is usually present. Leukopenia is generally noted. Patients are probably infectious during the prodrome and during the eruption.

### Treatment

- A Specific Measures None
- B General Measures Aspirin for malaise if required
- C Treatment of Complications
  - 1 Fetal abnormality is frequently found if the disease occurs during the first or early in the second trimester of pregnancy (See Prophylaxis)
  - 2 Encephalitis (code No 930 165) and thrombocytopenic purpura (code No 516 165) are very rare. Treat symptomatically
  - 3 Secondary streptococcal infection may occur and should be treated with penicillin (see # 459)

### Prophylaxis

Pregnant women who have been exposed to rubella may be given 5 to 20 cc. of immune serum globulin (gamma globulin) I M in an effort to prevent or modify the disease

## VARICELLA (Chickenpox) (code No 010 161)

Varicella is an acute communicable disease caused by a virus akin to that of herpes zoster. It is characterized by the eruption of crops of skin lesions

### Diagnosis

The incubation period is 2 to 3 weeks (usually 17 days)

Prodromal symptoms are usually slight and last only one day. Lesions erupt in crops and progress through the maculopapular, vesicular and pustular stages to crusts in about 3 days. The eruption is usually centripetal in distribution. The patient is infectious for one day before the onset and for 5 days thereafter. The late crusts may also occasionally be infectious.

#### Treatment

A Specific Measures None available

B General Measures

- 1 Isolate until primary crusts have disappeared
- 2 Bed rest until afebrile
- 3 Cleanliness of skin by frequent tub baths or showers when afebrile
- 4 Calamine lotion locally and antihistaminic orally may relieve the pruritus

C Treatment of Complications

- 1 Secondary bacterial infection of the lesions may be treated with bacitracin, tyrothricin or penicillin of optimum potency locally. If extensive penicillin is more likely to be given.
- 2 Post variella encephalitis may be treated only symptomatically.

#### Prophylaxis

The primary passive protection regularly follows the administration of 20 cc of convalescent serum which is rarely warranted.

### SMALLPOX (Variola) (code No. 010 176)

Smallpox is a serious communicable febrile disease characterized by rapid onset of constitutional symptoms followed by an eruption most marked on the face and extremities and often involving the mucous membranes.

The prognosis is extremely variable and depends on several factors. Previous effective vaccination prevails as a modifying factor. In cases with high fever and in confluent and hemorrhagic types of smallpox the prognosis is poor. The influence of the virus in the lesions is quite variable. If complications are present the prognosis is worse. The amount of scarring is variable but is more marked with secondary infection.

#### Diagnosis

The incubation period is 7 to 21 days. The prodromal illness lasts 2 to 4 days and consists of fever, prostration and headache and malaise, prostration and often vomiting, sore throat and cough. The onset of the eruption may be accompanied by temporary drooping of the voice. Maculopapules are preceded by shallow papules which become vesicles in about 3 days. On about the sixth day of eruption, pustulation occurs followed by crusting after the tenth day. Lesions are centrifugally distributed and are most dense on the face and distal part of the extremities. A recent successful vaccination usually precludes the diagnosis of smallpox. In a suspected case, infectivity is prevented from just before the onset until the late crust is shed.

Treatment

- A Specific Measures None
- B General Measures a Penicillin has a generally favorable effect probably due to control of secondary invaders which are almost an integral part of the disease
- C Local Measures
- 1 Mucous membranes Early in the disease provide good oral hygiene (see p 8 ) and apply petrolatum or mineral oil swabs to the nares
  - 2 Skin Gentle cleansing If lesions are confluent and suppurating treat as pyoderma (see p 85) Avoid itching by use of antipruritics (see p 68) restraints and sedation may be necessary
- D Treatment of Complications Treat as indicated for secondary infections otherwise treatment is symptomatic Complications include secondary infections of the skin mucous membranes and respiratory tract septicemia nephritis myocarditis and various neurological manifestations

Prophylaxis

Vaccination (see p 493)

### EPIDEMIC PAROTITIS (Mumps) (code No 621 170)

Mumps is an acute infectious disease caused by a specific virus which most commonly involves the salivary glands but frequently produces meningoencephalitis orchitis pancreatitis and oophoritis The prognosis is almost always favorable Testicular atrophy usually unilateral may follow orchitis but rarely produces sterility

Diagnosis

The incubation period is 2 to 4 weeks (usually 12 to 21 days) Swelling of the parotid or other salivary glands is the commonest manifestation and is rarely accompanied by severe systemic manifestations Headache and drowsiness abdominal pain and testicular pain and swelling usually associated with fever generally denote meningoencephalitis (confirmed by lumbar puncture) pancreatitis and orchitis respectively Complement fixing antibodies appear during convalescence Mumps is probably infectious just before the appearance of swelling and until swelling disappears

Treatment

- A Specific Measures None available
- B General Measures
- 1 Isolate until swelling is gone
  - 2 Bed rest during febrile period
  - 3 Aspirin or codeine for analgesia if required
  - 4 Alkaline aromatic solution mouth washes
  - 5 Mumps convalescent serum 20 cc mumps convalescent gamma globulin 2.5 cc I M or stilbestrol 2 to 5 mg (1/30 1/12 gr ) daily may reduce the incidence of orchitis in adult males
- C Treatment of Complications Complications are really less

common manifestations of the disease and not true complications. They may precede or occur in the absence of parotitis.

- 1 Meningoencephalitis (code No 912 170) May be a symptomatic
  - a Analgesics as necessary
  - b Lumbar puncture if necessary to reduce headache
- 2 Orchitis (code No 755 170)
  - a Suspension of scrotum in suspensory or towing bridge and application of ice bags
  - b Incision of tunica may be necessary in severe cases
  - c Codine or morphine as necessary for analgesia
  - d Injection of spermatic cord at external inguinal ring with 10 to 20 cc (2½ to 3 cc) of 1% procaine solution
- 3 Parotitis (code No 890 170) Symptomatic relief only  
Paracervical fluid if necessary
- 4 Oophoritis (code No 788 170) Symptomatic treatment only

### Prophylaxis

- A Mumps convalescent serum 20 cc (5 drops) 1 M may reduce incidence in exposed susceptible
- B Mumps virus vaccine may produce temporary active immunity  
Intradermal injection of virus antigen develops immunity if followed by local erythema

## **POLIOMYELITIS (Infantile Paralysis) (code No 972 171)**

Acute anterior poliomyelitis is an infectious disease of viral origin involving the central nervous system and manifested by muscle spasm and in many cases weakness. The overall mortality rate is 5 to 10 per cent. Almost all deaths occur in patients with bulbar involvement. Recovery of motor power is unpredictable and improvement may continue for months or years.

### Diagnosis

The incubation period is 3 to 21 days (usually 7 to 10 days). Pain is the commonest symptom (backache, backache, stiffness, soreness in the extremities, abdominal pain or sore throat). This is usually associated with fever of 3 to 5 days duration. Muscle weakness may appear at any time during the febrile period. The cerebrospinal fluid usually contains an increased number of lymphocytes but sugar and chloride content are normal.

Infectivity is greatest during the first week of the disease and possibly may be present before the onset, suggested from the presence of virus in the nasopharynx. Virus occasionally persists in the stools for many weeks.

### Treatment

- A Symptomatic None. Serum and plasma have never proved of benefit in controlled series
- B General Measures
  - 1 Pain
    - a Hot wet packs wrung dry to area of pain & to muscle spasm 2 to 3 times daily
    - b Aspirin or codeine as necessary for pain unrelieved by packs

- 2 Muscle spasm Hot packs neostigmine and curare and curare like drugs are of questionable value Muscle stretching after subsidence of the acute process should be carried out cautiously
- 3 Prevention of deformity and restoration of motor power Weak muscles should be supported by rolled towels and bags or light removable splints where necessary to prevent deformity Passive movement to the point of pain of all involved areas should be carried out daily as soon as fever & aides to prevent contracture and minimize atrophy As motor power returns active movement of involved areas should be carried out (under careful supervision) once or twice daily to prevent incoordination Pool therapy may allow reeducation of extremely weak muscles by minimizing the effect of gravity Rapid return of motor power usually occurs for about 3 months Following this period an increase in motor power may develop from exercises against resistance or use in ordinary activities as such as eating walking and climbing Braces may be required to support very weak muscles while somewhat stronger muscles are being used actively Canadian (short) clutches and spring suspension slings for the extremities also may aid in muscle reeducation Complete rehabilitation within the permanent limitation of the patient should be attempted so that the patient may resume an approximately normal life as early as possible At all times deformity should be avoided by appropriate supervision

#### E Treatment of Complications

- 1 Urinary retention requires repeated catheterization or an indwelling catheter Sulfonamide prophylaxis should be used
- 2 Paralysis of deglutition complicated by accumulation of secretions must be treated vigorously but carefully to prevent aspiration and atelectasis as well as hypoxia Elevation of the foot of the bed and careful repeated aspiration deep in the respiratory tract with mechanical suction should be utilized primarily When these measures are unsuccessful tracheotomy should be performed and suction carried out through the tracheotomy opening Penicillin should be used to prevent secondary infection
- 3 Paralysis of intercostal muscles and diaphragm requires the use of the respirator Attempt to wean the patient away from the respirator should be begun as soon as any respiratory involvement is observed and should be persisted in at whatever pace is possible The rocking bed and chest respirator should aid in the transition If the vital capacity diminishes or fails to increase return to more mechanical respiratory aid may be necessary to avoid fatigue of weak respiratory muscles

Oxygen may be given by nasal catheter or mask when hypoxia is evident by clinical observation or oximeter readings The negative pressure of the respirator and the rate of respiration should be adjusted according to need but should be the minimum required to insure adequate oxygenation

- 4 Respiratory center involvement (acute bulbar poliomyelitis code No 957 171) Patients whose respiratory

difficultly due to involvement of the respiratory center are generally not helped by the respiratory. Oxygen should be administered to prevent hypoxia.

- 5 Cardiovascular. Hypertension, hypotension and tachycardia may occur due to involvement of circulatory centers by poliomyelitis virus or hypoxia damage. Treatment is unsatisfactory. Oxygen should be used.
- 6 Atelectasis and pneumonia refer to causes of death in patients with respiratory paralysis and may be avoided by spirators or bronchoscopic antiseptic therapy.

### Polyphaxia

- 1 highly controversial subject at present.
- A Gamma Globulin. Given in doses of 0.2 cc per pound of body weight gamma globulin is said to be 88 per cent effective against paralytic effect of disease in a 3 week period and 75 per cent effective for 3 to 8 weeks (W. M. Hammon). Evidence would suggest that gamma globulin may have preventive or modifying effects on the incubation period.
  - B Salk Vaccine. A nationwide controlled vaccination program currently in progress will indicate the value of this vaccine.

## PSITTACOSIS (code no 910 173)

Psittacosis (ornithosis) is characterized by pneumonia, often migratory, usually associated with weakness. A history of contact with pet psittacine pigeon or rarely other birds is usually obtainable. Diagnosis is proved by isolation of virus from blood or sputum of the patient or by rising titer of complement fixing antibodies. Human to human transmission is rare although isolation precaution is recommended.

### Treatment

- A Spectinomycin.
  - 1 Oral. Spectinomycin (A. roemycin®) 0.5 Gm every 4 hours orally at 0.5 Gm 1/4 every 12 hours for 10 to 14 days.
  - or 2 Parenteral (aqueous) 100,000 units 1 M every 1 hour for 1 week.
  - or 3 Oxytetracycline (T. roemycin®) 0.5 Gm every 6 hours orally.
- B General Measures. Oxygen and sedation as required.

## ENCEPHALITIS (code no 930 1 )

The pathogen may be arthropod borne (East and West Nile virus, Japanese encephalitis, St Louis or Japanese B) post-infectious (mumps, varicella, etc.) or of unknown type (von Economo's etc.). Serological depression and focal abnormalities signs of meningeal irritation and convulsions may be noted. Lymphocytosis is found in the cerebrospinal fluid and correlated with the rise of sugar in the liquor. Pathogenicity of virus is indicated by the isolation and re-inoculation of the arthropod borne types and in mouse encephalitis or atypical.



## 456 Dengue Rabies

### Treatment

A Specific Measures None

B General Measures

- 1 Repeated lumbar punctures may relieve symptoms
- 2 Prevention of decubiti pneumonia and urinary tract infections is important
- 3 Anticonvulsants as needed (see pp 351 and 533)

## LYMPHOCYTIC CHORIOMENINGITIS (code No 910 160)

Lymphocytic choriomeningitis is a viral infection of the central nervous system which is clinically indistinguishable from non paralytic poliomyelitis or mild encephalitis. The virus may be isolated from the blood or spinal fluid or diagnosis may be confirmed by a rising titer of neutralizing or complement fixing antibodies. Incubation period is probably 8 to 21 days.

### Treatment

A Specific Measures None

B General Measures As in encephalitis (see above)

## DENGUE (code No 010 162)

Dengue is an acute infectious disease caused by a virus transmitted by mosquitoes. The incubation period is usually 5 to 8 days following the bite of an infected Aedes mosquito. Onset is with chilliness aching of head back and extremities anorexia and severe prostration. Conjunctival injection and generalized lymphadenopathy may be found. The fever usually lasts 5 to 6 days and may be of the saddle back form. A scarlatiniform or maculopapular eruption occurs on the third to fifth days and lasts up to 3 days. Leukopenia is usually marked. Fatality is extremely rare.

### Treatment

A Specific Measures None

B General Measures

- 1 Salicylates or codeine as required for discomfort
- 2 Gradual restoration of activity during prolonged convalescence

### Prophylaxis

A Control of mosquitoes by screening and DDT

B Dengue vaccine shows promise experimentally

## RABIES (code No 010 174)

Rabies is an acute viral infection primarily of animals which is occasionally transmitted to man. It is characterized by apathy or hyperexcitability paralysis and invariably results in death.

### Diagnosis

The incubation period is usually 2 to 3 weeks (occasionally as

long as 1 y r) f Losing the bit of a rabid animal Onset occ s with pain and o mbo as at the ait of i oc lati n followed by de pression irritability ad mild dysphagia This is followed by hyperaheia and m aci spasms partic ularly of the pharynx E vtuallu paralysis and death occur

### Tx Im i

A Specific Therapy None

B General

- 1 Absolute quiet and freedom from stimulation
- 2 Sedation as indicated for preventing convulsions

### Prophaxis

A Quarantine of animal producing bite

B Ca i rization of wound with f ming nitric acid followed by a trill ation of th e id with lime water or the o gh w shing with green soap

C Rabies vaccine 2 cc subcut daily f 14 days following positive diagnosis of rabies or following bite by a suspected animal if a im i cannot be observed or if bite is on the head

## YELLOW FEVER (code No 010 170)

Y low fever is an te infection of man nd monkeys due to a virus trans mitted by A des mosquitoes It is characteriz d by fever r lative bradycardia icteru and hemorrhagic phenomena The mortality rate of y low f e is q it v et ble including mited It is probably 5 per ce t

### Incubal

The incubation period is 3 to 6 days The on t is with chill he do be and headache Th i et oft de lines i mporarily af 3 days during which time th patient i f bed and toxic and may have a very na se and vomiting The conjunctiva are injected and the tongue red This is follow d by pallor c hymosa ill dig sm black vomit Light jaund. met na albuminuria and proteinuria Le kape is ad relative bradycardia e usually e en

### Treatment

A Specific Treatment None

B General

- 1 Liquid diet Limit food to high-carbohydrate high protein liquids e to, cat d
- 2 Intravenous glucose and saline as required
- 3 Analgesic and sedatives as necessary
- 4 Saline enemas for abdominal pain

### Prophaxis

A Vaccination control by adequate screening use of DDT

B Treatment of the mosquito by

## DISEASES DUE TO RICKETTSIAE

The rickettsiae are arthropod borne organisms which produce widespread nodular thrombotic and necrotic lesions in the smaller blood vessels and capillaries. The severity of involvement varies with the different species of the rickettsia. The seriousness of the prognoses of epidemic typhus, Rocky Mountain spotted fever and scrub typhus has been greatly reduced since the advent of specific antibiotic therapy.

Diagnosis

- A Typhus Fever (code No. 010 184)** Incubation period 6 to 15 days. Typhus fever is caused by *Rickettsia prowazekii* (epidemic type) or *Rickettsia mooseri* (murine type). The former is transmitted by body lice, the latter by rat fleas. The diseases are similar except that the murine variety is less severe. Onset is abrupt with chills, fever, aching and prostration. Delirium, stupor or coma may occur. A macular, papular or hemorrhagic rash begins on the fourth to seventh days, appearing first on the trunk and spreading to the extremities, usually sparing the face, palms and soles. Leukopenia occurs early. Diagnosis may be confirmed by complement fixation reaction or Proteus OX 19 agglutinins appearing during the second week.
- B Rocky Mountain Spotted Fever (code No. 010 181)** The incubation period is 3 to 14 days. Rocky Mountain spotted fever is caused by *Rickettsia rickettsii* and is transmitted by tick bite (principally *Dermacentor andersoni* and *D. variabilis*). Prodromal symptoms of chilliness, anorexia and malaise may occur. The onset of chills, fever, headache, photophobia, pain in the extremities is usually sudden. A red, later dusky or hemorrhagic maculopapular rash appears first on the wrists and ankles from the second to sixth days and extends rapidly over the entire body including the face, palms and soles. Leukopenia is usually present early. Complement fixing antibodies and agglutinins for Proteus OX 2 or OX 19 appear during the second week.
- C Scrub Typhus (Tatsugamushi Fever) (code No. 010 183)** Scrub typhus is caused by *Rickettsia tsutsugamushi* (*R. tsutsugamushi*) and is transmitted by larval mites. An eschar at the area of inoculation is common. The onset is sudden with chills, fever, malaise and cough. A dull red maculopapular eruption appears on the trunk from the fifth to eighth days and may extend to the extremities. Specific complement fixing antibodies or Proteus OX K agglutinins appear during the second week.
- D Q Fever (code No. 010 185)** Q fever is caused by *Coxiella burnetii* and is apparently acquired from sheep, goats and cattle in a manner not yet determined. Headache, fever, cough and stiff neck are common symptoms. Repeated rigors may occur. Pneumonitis or hepatitis may be demonstrated. Specific complement fixing antibodies appear during the second or third weeks.
- E Rickettsial Pox (code No. 010 187)** Rickettsial pox is an infection caused by *Rickettsia akari* introduced by the bite of a mite. A lesion which passes through the stage of a pleomorphic vesicle and

es har pre dex the n i of fev r chills h adache ph to  
phobi and muscula soreness by about a we k A generalized  
rs h which evolves through papular icular and crusting  
stag a ppears at the onset of fever or a few days later  
Le k penia usually is present

- Tre im f M sur a  
A Sp f Ail x is d seases  
a Chlor tacycll (Aureomy 1 0) 0.5 1.0 Gm orally  
very 6 hours for 3 to 7 days r 0.5 Gm 1 V every  
12 hours  
or b Oxytetracycline (Terramycin) 0.5 Gm orally ev ry 6  
hours for 3 to 7 days or 1 Gm 1 V every 12 hou s  
or c Chl romph nic 1 U S P (Chl romycetin) 0.5 Gm  
orally every 6 hours for 2 to 7 day  
er 2 Typhus nd Rocky Mountain spotted f ers Par amino  
benzoic acid 1 0 2 2 Gm (Kg body wt (0.5 1.0 Gm /lb )  
orally per d y in divid d do es every 4 hours for s eral  
days till r cess tion of f er (for typhus and Rocky Mountain  
spotted f vers)  
D Ce ral M rre  
1 P ent ral D de oxyge and sedation s req ired  
2 Cibe s pporil means es as needed  
3 Delousing proc dures m et be t ried out f r louse borne  
inf tions ( s p 93)

- Proot la is  
A Soc r M s  
1 3-4 day interval  
2 Rocky Mount in spotted fever vs cine 1 0 c subcut 3  
tim s at 5 to 7 day intervals  
B (General) M Delou 1 g is very important in louse borne  
r muc typh (s p 93)

## DISEASES DUE TO BACTERIA

### SCARLET FEVER (code No. 010 102) and STREPTOCOCCIC SORE THROAT (code No. 621 102)

Sc let f ver and streptococcic sore throat (follicula tonsil  
lyc s repharct (Lanc fl d Group A) s by 8 hemo  
tion stain man-entator due to erythrogenic toxin are present  
brun pny the re s

The mortality rat from streptococci sore throat and t r l i  
frve in the Ent d Stat is 0.5% or 1 s Rhe matic fever is pro  
bely more common than general y app ealed and may be app  
at only when serial betrocra diagrams are tak n d ring coova  
bruce

Diagnosis

The incubation period is 2 to 7 days. The onset is usually abrupt with chills, fever, headache, pain in the extremities or abdomen, vomiting and sore throat. The throat is usually fiery red and moderately edematous. Exudate, if present, consists of patches of whitish material which may be easily wiped off. The rash of scarlet fever consists of a punctate erythema which is densest in the skin folds of the axilla and groin but does not appear on the extensor surfaces of the upper extremity. Strawberry tongue and stippling of the soft palate may be noted. Diagnosis may be confirmed by culture. Sedimentation rate is increased and leukocytosis is present.

The duration of the infection varies; it may be prolonged by a convalescent carrier state.

TreatmentA. Specific Measures

1. Penicillin procaine 300 000 units daily I M. Penicillin must be continued 5 to 7 days or relapse may occur. Aqueous penicillin 30 000 to 40 000 units I M every 2 hours may be used or oral penicillin 200 000 units every 4 hours. Local penicillin by lozenges is worthless.
- or 2. Chlorotetracycline (Aureomycin®) 0.2 to 0.5 Gm every 6 hours, oxytetracycline (Terramycin®) 0.5 Gm every 6 hours or erythromycin 0.2 to 0.5 Gm every 6 hours are effective but may be followed by bacteriological or clinical relapse.
3. Sulfonamides have no effect on the course of streptococcal sore throat or scarlet fever but may prevent complications if given for 2 weeks. Dosage 0.5 Gm (7½ gr) every 4 hours with equal or double quantities of sodium bicarbonate.
4. Scarlet Fever Streptococcus Antitoxin (9 000 to 100 000 units) may be given I M with benefit in severely toxic cases of scarlet fever.
5. Convalescent serum 25 to 150 cc (1 to 5 oz) may be used similarly to antitoxin and may be given I V.

B. General Measures

1. Bed rest until afebrile and sedimentation rate is normal.
2. Diet as suited to soreness of throat.
3. Hot saline or 30% glucose gargles or throat irrigations 3 or 4 times daily for relief of sore throat.
4. Aspirin or codeine as necessary for symptomatic relief.

C. Treatment of Complications

1. Complications due to infection include cervical adenitis, rhinitis, sinusitis, otitis, mastoiditis, pneumonia, empyema, septic arthritis, and septicemia. Treatment with penicillin is usually effective (see p. 502).
2. Complications of unknown etiology
  - a. Rheumatic fever may be prevented by early vigorous treatment of the infection with penicillin (see p. 518).
  - b. Acute hemorrhagic glomerulonephritis (see p. 293).

3. Treatment of Carriers 300 000 units of penicillin procaine complex daily I M for 6 days usually abolishes the carrier state.

Prophylaxis

- A. Serum Tilet toxin in 5 weekly injections of 300 2000 3000 10 000 and 10 000 units as best prophylaxis the toxic manifestations of a latent diphtheria or prophylaxis streptococcal infection
- B. Penicillins 0.5 Gm (1½ gr) b.i.d. penicillin 100 000 units by mouth b.i.d. or benzathine penicillin 600 000 units i.m. once a month reduce the incidence of streptococcal infection. These should be reserved for individuals with rheumatic lesions to prevent recurrence of rheumatic fever

**DIPHTHERIA (Pharyngeal code No 631 125)**

(Laryngeal code No 330 125)

(Nasopharyngeal code No 318 125)

Diphtheria is an acute infectious disease caused by *Corynebacterium diphtheriae* and characterized by the formation of pseudomembrane at the portal of entry usually the respiratory tract and by the edibility of exotoxin at distant sites

The mortality rate of diphtheria generally varies between 10 and 30 per cent. Older individuals do poorly and delay of treatment results with a high mortality rate. Myocarditis appearing early is frequently fatal and disturbances of conduction or the appearance of rapid arrhythmias imply a relatively poor prognosis. However if the patient survives recovery is usually complete. Death is rarely fatal unless paralytic complications of cranial nerves and intercostal muscles are paralyzed. Surviving patients recover slowly but completely

Diagnosis

The incubation period is 2 to 7 days. Symptoms depend on the site of the lesion but include sore throat, nasal discharge or hoarseness accompanied by malaise and low grade fever. The pseudomembrane is typically greyish, homogeneous and tenacious. Edema and a raw zone of epithelium around the lesion are usually found. Diagnosis is confirmed by culture. Latent disease must be excluded as long as C. diphtheriae persists in the nasopharynx the carrier state is not uncommon.

TreatmentA. Supportive B. Specific

1. Supportive Treatment must be given in all cases where diphtheria is suspected by simple clinical examination. The intravenous route is preferable in the treatment of the mild cases or in those who are unable to tolerate oral. Conjugate and A. tests for serum sensitivity (see p 494) should be done in all cases and administration (see p 494) started as soon as any. The dose varies with the duration of the disease and the location of the lesion and the site of the portal. A large dose should suffice.

## DOSE SCHEDULE

Location	Child	Adult
Anterior nasal	5000 units	10,000 nits
Mild pharyngeal	10,000 nits	20,000 units
Moderate pharyngeal	20,000 units	40,000 nits
Severe pharyngeal and nasopharyngeal	40,000 units	80,000 units
Laryngeal	10,000 nits	20,000 units
Any two sites or late cases	40,000 units	80,000 units

- 2 Penicillin procaine 300,000 units daily or penicillin 50,000 units every 3 hours accelerates slightly the disappearance of the organism from the throat and acts against secondary streptococcal invaders. It does not alter the course of the diphtheria itself.

## B General Measures

- 1 Absolute bed rest for at least 3 weeks and until Ecg is normal
- 2 Liquid to soft diet as tolerated
- 3 Hot saline or 30% glucose throat irrigations 3 or 4 times daily
- 4 Aspirin or codeine as required for pain

## C Treatment of Complications

- 1 Myocarditis (code No 430 125 9) This may occur at any time up to several weeks after onset and may be associated with peripheral vasomotor collapse. Anginal or abdominal pain, nausea and vomiting or syncope may be noted. Deterioration of the mitral first heart sound, drop in blood pressure, gallop rhythm or any arrhythmia may be found. Ecg evidence is usually demonstrated in serial records.
  - a No definitive treatment is known
  - b Oxygen by tent or mask may be needed
  - c Hypertonic glucose solution 100 cc of 20% solution daily may aid
  - d Digitalis and quinidine should be reserved for rapid arrhythmias
- 2 Neuritis (code No 98 125 9) generally does not begin until at least 3 weeks after the onset. Nasal voice, regurgitation of fluids through the nose (N IX), paralysis of accommodation (N III), dysphagia and dysphonia (N X) and rarely involvement of the extremities which is associated with paresthesias, weakness and depression of reflexes. Nasal feeding should be attempted in such cases. Corrective splinting and physical therapy may be of aid.
- 3 Respiratory tract obstruction. Croupy cough, stridor and dyspnea suggest laryngeal obstruction.
  - a Suction of membrane and secretions under direct laryngoscopy may help
  - b Intubation or tracheotomy should be performed before the appearance of cyanosis if the distress increases

## D Treatment of Carriers. Penicillin has very limited effect on the carrier state.

# Prophylaxis

- 1 Children Three injections (0.5, 1.0 and 1.0 cc) of diphtheria toxoid (formalin alumina aluminum hydroxide precipitated) at one month intervals during the first 6 months of life (may be combined with tetanus toxoid and pertussis vaccines). Follow by Schick test in 3 to 6 months. Give 1.0 cc booster at 2 years and at start of school.
- 2 Adults
  - 1 Sensitivity test. Maloney's test for sensitivity to old 0.1 cc of 1:20 dilution of plain toxoid intradermally. Read like Schick test at 24 to 48 hours.
  - 2 If Maloney test is negative proceed with a live attenuated pertussis vaccine.
  - 3 If Maloney test is positive give 0.1 cc of 1:10 dilution of formalin toxoid intracutaneously at 3 week intervals.

## PERTUSSIS (Whooping Cough) (code No. 350.108)

Pertussis is an acute communicable infection of the respiratory tract. It is caused by *Bordetella pertussis* and is characterized by paroxysmal cough, whoop, and subacute lymphocytosis. Until recently the mortality rate of pertussis in infants and young children was 20 per cent. This has been materially reduced with modern antibiotic therapy. Older children rarely die of pertussis.

### Differential

The incubation period is 7 to 16 days. The onset is with coryza followed by gradually increasing cough. After 1 to 2 weeks the cough becomes paroxysmal, especially at night, and is often followed by a whoop. Infrequent in young infants. Absolute lymphocytosis appearing during the paroxysmal stage. The diagnosis may be confirmed by cough plate or nasopharyngeal swab cultured on Bordet-Gengou's medium. It is a negative test early in the disease and decays as until the organism disappears from the nasopharynx in about one month.

### Treatment

- 1 Antibiotics
  - a Chloramphenicol (Chloromycetin®) 25 mg/Kg (11 mg/lb) per day orally
  - b Oxytetracycline (Terramycin®) 50 mg/Kg (23 mg/lb) per day orally
  - c Streptomycin 1.0 Gm per day in divided doses i.m. if one or 2 may be effective
  - d Chloramphenicol U.S.P. (Chloromycetin®) 50 mg/Kg (23 mg/lb) per day orally
  - e Polymyxin B 3.0 mg/Kg (1.4 mg/lb) per day i.m. is also promising but this drug may be too toxic (ref p. 510)
- 2 Hyperimmune serum or hyperimmune gamma globulin may be used in severe cases and reduce mortality. 20 hyperimmune serum or 3.3 cc hyperimmune gamma globulin given daily or every other day i.m. for 3 days.



**C Complications and Treatment**

- 1 Myocarditis and renal failure are common in severe cases  
No specific treatment is available
- 2 Cranial nerve lesions (especially N VIII) are usually permanent
- 3 Arthritis requires no treatment other than paracentesis for relief of pain

**Prophylaxis**

total of 1.0-2.0 Gm (15-30 gr) sulfadiazine orally to exposed persons or carriers in two doses taken the same day

**PNEUMOCOCCIC MENINGITIS (code No 910 101)**  
**STREPTOCOCCIC MENINGITIS (code No 910 102)**  
**STAPHYLOCOCCIC MENINGITIS (code No 910 103)**

Symptoms are similar to those of meningococcic meningitis but a preceding infection is usually present and a focus is often demonstrable in the lungs (pneumococcic) the middle ear or sinuses. The spinal fluid must be cultured and examined to determine the causative agent.

**Treatment****A Specific Measures**

- 1 Treat as meningococcic meningitis (see above) and also give 10,000 units of penicillin in 10 cc physiological saline intrathecally once daily until CSF glucose is normal
- or 2 Aqueous penicillin 1,000,000 units I.M. every 2 hours
- 3 Erythromycin 0.5 Gm every 6 hours for staphylococcic meningitis
- 4 Oxytetracycline (Terramycin®) and chlortetracycline (Aureomycin®) may also be effective do not combine with penicillin

**B General Measures** Symptomatic and supportive**HEMOPHILUS INFLUENZAE MENINGITIS (code No 910 110)**

This form of meningitis may develop gradually or suddenly. It usually occurs in infants less than 2 years old. The symptoms are similar to those of any purulent meningitis (see p. 463).

**Treatment****A Specific Measures**

- 1 Dihydrostreptomycin (adults 1.0 Gm, children 250 mg) I.M. every 6 hours for one week and streptomycin 25 mg in 10 cc physiological saline solution intrathecally daily until cerebrospinal fluid glucose content is normal
- 2 Severe cases should also be given sulfadiazine-sulfamerazine or a mixture of equal parts of each 150 mg/kg body wt (65 mg or 1 gr/lb) per day. Give equal or double doses of sodium bicarbonate.
- 3 Hemophilus influenzae antiserum (Type B) should also be given to patients with delayed response 25 to 100 mg of antibody nitrogen daily I.V. until a serum produces quelling reaction.

- 4 Chloro-tracycline (Aureomycin®) 0.5 Gm. every 6 hours is of value. Oxy-tracycline (Terramycin®) and chloramphenicol (U.S.P. Chloramycetin®) are also effective.
- D. General Measures. Treat symptoms as they arise and maintain good nutrition and adequate fluids.

### TUBERCULOUS MENINGITIS

(Leptomeningitis code No 912 123)  
(Pachymeningitis code No 911 123)

This disease is caused by spread of the tubercle bacilli from a focus usually in the lungs or in the peritracheal peribronchial or mesenteric lymph nodes. There is usually a gradual onset of symptoms with loss of consciousness, anorexia and fever followed by headache, vomiting, night terrors, convulsions and rigidity of the back, opisthotonos, paralysis and coma.

Cerebrospinal fluid frequently xanthochromic, is under increased pressure and on standing may form a web and pellicle from which the organisms may be demonstrated by smear culture or guinea pig inoculation. Cells and protein are increased but sugar and chloride are decreased.

#### Treatment

- A. Specific Measures.
- 1 Streptomycin 30 mg./Kg. body wt. and dihydrostreptomycin 30 mg./Kg. body wt. per day in divided doses every 6 to 12 hours for 3 months.
  - and 2 Streptomycin 3 mg./Kg. of body wt. (1 mg./lb.) intrathecally daily for 2 weeks every other day for 2 weeks and twice a week for 2 weeks.
  - and 3 Para-aminosalicylic acid 3 to 5 Gm. every 8 hours by mouth or sodium para-aminosalicylate 15 to 30 Gm. daily for one year.
  - and 4 Isonicotinil hydrazide (isoniazid, INH) 3 mg./Kg. body wt. per day divided into 2 or 4 doses for one year.
- B. General Measures. Treat symptoms as they arise and maintain good nutrition and adequate fluids.

### TYPHOID FEVER (code No 910 115)

Typhoid fever is an infection caused by *Escherichia typhosa* and characterized by the terminal ulceration of the lymphoid tissue of the small intestine and generalised toxemia. The mortality of typhoid fever varies from 3 to 25% following individuals do poorly.

#### Disease

The incubation period is 3 to 11 days. A gradual onset of fever and malaise are associated with cough or epistaxis (a) followed by a period of 3 or more weeks of sustained fever and then gradual defervescence. Rose spots, prostration, gurgly stools, tachycardia, delirium and prostration may be noted. Leukopenia is the rule. Lesions of the organs are from the blood, stool or urine or a high percentage of agglutinins confirm the diagnosis.

## 470 Tetanus

- or 2 Chlorotetracycline (Aureomycin®) orally Give 100 mg once the first day 50 mg twice the second day 50 mg 3 times the third day and 0.5 to 1 Gm every 8 hours for the following 12 to 14 days Small initial dosage avoids Herxheimer like reaction
- or 3 Chloramphenicol U S P (Chloromycetin®) 60 mg /Kg body wt (27 mg /lb) orally initially then 0.25 Gm every 3 hours until afebrile 7 days
- or 4 Oxytetracycline (Terramycin®) 0.5 Gm every 6 hours for 2 weeks
- or 5 Dihydrostreptomycin 0.5 Gm 1 M every 8 hours for 2 weeks and sulfadiazine sulfamerazine mixture 3 Gm (45 gr) initially and 1 Gm (15 gr) every 6 hours for 2 to 3 weeks

### III General Meas res.

- 1 Bed rest during acute febrile stage
- 2 High vitamin intake

### Prophylaxis

- A Destruction of infected dairy animals and immunization of susceptible animals
- B Pasteurization of all milk and milk products

## GAS GANGRENE (code No 12)

Gas gangrene due to any of several anaerobic Clostridia is an occasional complicating infection of wounds which are soiled with dirt or feces Devitalization of tissue is usually present Fever chills and local swelling are usually seen Gas bubbles in the tissues may be demonstrated by x rays Bacteremia may be present Anaerobic cultures of discharge from the wound or blood confirm the diagnosis

### Treatment

- A Specific Measures
  - 1 Penicillin 100 000 units 1 M every 3 hours
  - and 2 Polyvalent gas gangrene antitoxin 20 000 units at once and repeat every 6 to 8 hours
  - and 3 Full doses of sulfadiazine sulfamerazine mixture (see p 499)
- B Surgical Measures Adequate surgical debridement and exposure of infected areas

## TETANUS (code No 010 11x)

Tetanus is a nervous system intoxication caused by fixation of Clostridium tetani toxin which enters through an open wound and infects injured tissues the disease is characterized by tonic contractions of striated muscle

### Diagnosis

The incubation period is 5 days to 3 weeks The first symptoms usually are pain and tingling at the site of inoculation which may be an insignificant wound that has become contaminated with

If this is followed by instability of the trunk muscles, stiff neck and extremities, and spasms of the abdominal muscles. Rigidity of the neck and trachea is a sardonicus stiff neck. Rigid abdominal muscles and hyperactive reflexes are found. Tonic convulsions gradually appear and are precipitated by slight stimulation. The mortality rate of the disease is approximately 40%. A long incubation period and the delayed appearance of tonic convulsions are favorable signs.

**Treatment**

Drug	Dose	Frequency	Notes
Abolite	100 mg	4 times daily	with minimum stimulation
Atropine	1-2 mg	4 times daily	if necessary
Barbiturates	100 mg	4 times daily	if necessary
Curare	100 mg	4 times daily	if necessary
Diaphragm	100 mg	4 times daily	if necessary
Emetine	100 mg	4 times daily	if necessary
Epinephrine	100 mg	4 times daily	if necessary
Hydrocortisone	100 mg	4 times daily	if necessary
Insulin	100 mg	4 times daily	if necessary
Iron	100 mg	4 times daily	if necessary
Penicillin	100 mg	4 times daily	if necessary
Physostigmine	100 mg	4 times daily	if necessary
Pyridostigmine	100 mg	4 times daily	if necessary
Quinine	100 mg	4 times daily	if necessary
Sedatives	100 mg	4 times daily	if necessary
Sodium Amytal	100 mg	4 times daily	if necessary
Strychnine	100 mg	4 times daily	if necessary
Thyroid	100 mg	4 times daily	if necessary
Veronal	100 mg	4 times daily	if necessary
Yodserol	100 mg	4 times daily	if necessary

**Prognosis**

The prognosis is generally good, but it is necessary to watch for complications. The mortality rate is approximately 40%. The disease is usually fatal within 10 days.

### BOTULISM (code No. 010 120)

Botulism is a food poisoning caused by the ingestion of the toxin of Clostridium botulinum. It is a rare disease, but it is fatal. The incubation period is usually 12 to 36 hours. The symptoms are usually double vision, drooping of the eyelids, and difficulty in swallowing. The disease is usually fatal within 10 days.

**Diagnosis**

The diagnosis is usually made by the presence of the toxin in the food. The toxin is usually found in the food. The disease is usually fatal within 10 days.

## 474 Relapsing Rat bite Fever

rest for one week should be continued for prolonged periods. Hemolytic anemia should be guarded against by frequent blood counts

or ☐ Isonicotinic acid hydrazide (isoniazid INH) 5 mg /Kg body wt per day in 3 or 4 doses

## DISEASES DUE TO SPIROCHETES

### RELAPSING FEVER

(Louse borne code No 010 1411)

(Tick borne code No 010 1412)

Relapsing fever is characterized by recurring febrile episodes of 3 to 5 days duration following a bite by an infected tick or louse. Diagnosis may be confirmed by demonstration of *Borrelia recurrentis* in the blood on direct examination or by animal inoculation

#### Treatment

##### A Specific Measures

1 Penicillin 50 000 units I M every 3 hours or penicillin procaine 300 000 units I M daily for 10 days

or 2 Chlorotetracycline (Aureomycin®) 0.5 Gm every 6 hours orally

or 3 Chloramphenicol U S I (Chloromycetin®) or oxytetracycline (Terramycin®) may be expected to prove effective

B Supportive and symptomatic measures as needed

### SPIROCHETAL JAUNDICE (Weill's Disease) (code No 010 142)

Spirochetal jaundice is characterized by severe constitutional symptoms purpuric skin lesions jaundice and nephritis. A history of contact with rats may be obtained (e.g. sewer workers warehousemen). Dark field examination of the blood or urine revealing the *Leptospira icterohaemorrhagiae* or a high titer of specific agglutinins confirms the diagnosis

#### Treatment

##### A Specific Measures

1 Chlorotetracycline (Aureomycin®) 0.5 Gm orally every 6 hours

or 2 Penicillin 100 000 units every 3 hours I M

B Supportive and symptomatic measures as indicated

### RAT BITE FEVER (Sodoku) (code No 010 134)

Rat bite fever is caused by *Spirillum minus* and is characterized by a recurrent chancre at the site of inoculation accompanied by regional adenitis fever and a macular rash. The fever is episodic and recurrent

#### Treatment

##### A Specific Measures

- 1 Penicillin 100 000 units every 3 hours I.M. or penicillin procaine 300 000 units I.M. every 12 hours
- 2 Chlorotetracycline (Aureomycin®) 0.5 Gm. every 6 hours
- 3 B 5 p.p.tive and symptomatic measures as indicated

### YAWS (Frambesia) (code No 010 140)

Yaws is an infectious disease produced by *Treponema pertenue* and characterized by granulomatous lesions of skin, mucous membranes and bone. Yaws is rarely fatal.

#### Diagnosis

Yaws is acquired by direct non-venereal contact. The mother's yaws is painless papules which later ulcerate, appears 2 to 4 weeks after exposure. It is to 12 weeks later similar secondary lesions appear and last for several months or years. Late gummatous lesions may follow. Visceral involvement is rare. The Wassermann and flocculation tests are positive and the spirochete may be demonstrated by dark field examination.

#### Treatment

- 1 Penicillin procaine 300 000 units I.M. daily for 7 to 10 days
- 2 Chlorotetracycline (Aureomycin®) 0.5 Gm. every 6 hours for 10 days
- 3 Dichlorophenarsone 40 mg. I.V. weekly for 3 to 6 weeks
- 4 General Hygiene, Chloroform of lesions to of at least 100 per cent

#### Prophylaxis

None other than good hygiene

## INFECTIOUS DISEASES OF UNDETERMINED ETIOLOGY

### VINCENT'S ANGINA (Stomatitis) (code No 010 141)

Stomatitis is an ulcerative infection of the oropharynx of doubtful etiology. Fusiform and spirochete infection and herpes may also have been incriminated.

#### Treatment

- 1 Penicillin (procaine) 300 000 units I.M. daily for 7 to 10 days (Probably against secondary infection)
- 2 Penicillin procaine 300 000 units daily I.M. in severe cases
- 3 Chlorotetracycline (Aureomycin®) 0.5 Gm. every 6 hours (Secondary infection or asymptomatic lesions may respond to tetracycline or erythromycin)
- 4 General Hygiene or hydrogen peroxide mouth washes
- 5 Correction of oral hygiene by a dentist

- 6 X rays may reveal alteration of contour of diaphragm hepatomegaly or right lower chest involvement
- 7 Material (anchovy paste like) may at times be aspirated from carefully localized abscess mass
- 8 Complement fixation test may help confirm diagnosis
- C Amebiasis of Other Organs Diagnosis is difficult and is possible only by maintaining a high index of suspicion of specific organ involvement (based on clinical manifestations) in patients with known or suspected amebiasis
- D Asymptomatic Amebiasis This diagnosis must be reserved for cases in which routine stool examination reveal cysts of *E. histolytica* but clinical findings (including sigmoidoscopic examination) are completely negative
- E Therapeutic Test A therapeutic trial of anti amebic drugs particularly chloroquine or emetine may be warranted
  - 1 If diagnosis is doubtful after careful investigation and amebiasis (especially hepatic) is clinically suspected
  - 2 If fulminant diarrhea is present and diagnosis is clinically suspected but cannot be established and no other organism can be found

### Treatment

#### A General Measures

- 1 Report case to local health authorities
- 2 Bed rest is required for certain patients those with frank dysentery hepatic or other non enteric involvement and all patients receiving emetine therapy (See below)
- 3 Diet
  - a If diarrhea is present follow the dietary measures as outlined for nonspecific diarrhea (see p 258)
  - b If there is hepatic disease follow the dietary measures outlined under the management of chronic hepatic disease (see p 280)
  - c Iron salts should be given if anemia is present (see p 219)

#### B Acute or Chronic Amebic Dysentery

- 1 Specific drugs In the presence of dysentery it is probably safer to assume that organisms have invaded extra intestinal tissues With this in mind an adequate course of therapy should include not only an agent effective against intestinal forms but also a drug which is effective in the extra intestinal tissues (see table below)

Effectiveness of Anti amebic Drugs

	Against Intestinal Organisms	Against Extra Intestinal Organisms
Chloroquine	±	+
Emetine	±	+
Carbarsone and Milidil®	+	
Vioform®	+	
Erythromycin	+	±
Fumagillin	+	±

Combinations of chloroquine (or em. use) and an asexual (Carber one or Milibis®) or an iodine-containing compound (Vioform®) are commonly employed. If the organisms prove resistant erythromycin or fumagillin can be tried. Dosages are given below.

- a. Chloroquine Diphosphate U.S.P. 0.5 Gm (1½ gr) (or 0.2 Gm of the base) b.i.d. for 3 days followed by 0.2 Gm (¾ gr) daily for 7 to 10 days.
- b. Em. use Hydrochloride Injection U.S.P. R.P. 65 mg (1 gr) daily subcut. for 6 days will control acute symptoms and eradicate infection in 15 per cent of cases. Em. use has been replaced by less toxic and equally effective agents such as chloroquine (see above).
- c. Calcium ion U.S.P. B.P. 0.25 Gm (3¾ gr) i.i.d. orally for 7 to 10 days or Bismuth Glycylarsenate N.N.R. (Milibis®) 0.5 Gm (1½ gr) i.i.d. for 7 days.
- d. Iodochlorohydroxyquinoline N.F. (Vioform®) 0.25 Gm (3¾ gr) i.i.d. orally for 14 days.
- Erythromycin 13 mg per Kg body weight daily for 10 to 14 days. This antibiotic is effective for the treatment of acute amoebiasis.
- f. Fumagillin 0.5 to 1 mg per Kg body weight daily for 10 to 14 days. Employed if drug refractive cases of chronic amoebiasis.

3. Evaluation of therapy. Following completion of therapy wait on stool and examine stools on three successive days. If still positive repeat above course of treatment and re-examine stool first course.
- b. If negative give a further course of treatment. Examine stool at 14 and 21 intervals until a total of 12 specimens have been found to be negative.

4. To the reaction of the organism to drugs. Em. use hydrochloride of Control material used in experimental disease as well as vomiting on a large scale which may result and protection on day or a. Special observation and precautions include the following:
  - (1) Bed rest without lavatory privilege.
  - (2) Stools are determined b.i.d.
  - (3) Pulse determination q.i.d.
  - (4) Daily examination of patient.
  - (5) Fasting before and after course of therapy.
  - (6) Withdraw drug in the event of toxicity.
- b. A. i. chemical compounds (Carbersone, Milibis®). Control material is sensitive to the use of these vomiting and diarrhoea has not been observed. Daily inspection for toxic symptoms is necessary. Stop drug in event of toxicity.
 

Iodoquinol (as compound) Iodochlorohydroxyquinoline (Vioform®) = antiamoebic (in experimental disease as well as in acute and chronic cases). Daily therapy for 1-2 weeks (uncommon) is necessary. Stop drug in event of toxicity and in 1-2 days.



## II Hepatic Amebiasis

### 1 Measures for hepatitis

- a Chloroquine Diphosphate N N R (Aralen®) is the drug of choice in hepatic amebiasis since it has proved to be effective in emetine resistant cases and is much less toxic. Like emetine this drug has rather feeble intestinal effects. It is therefore necessary to follow the course of chloroquine with Vioform® carbarsone or one of the antibiotics notably erythromycin or fumagillin (see above).  
**Dosage:** Chloroquine diphosphate 0.5 Gm (7½ gr) (or 0.3 Gm of the base) b i d for 2 days followed by 0.5 Gm (7½ gr) daily for 12 to 14 days.
- or b Emetine hydrochloride injection 80 mg (1 gr) subcut daily for 9 days. If chloroquine is not available or fails to provide a desirable therapeutic effect.
- c Hepatic function tests may determine degree of liver involvement.
- d Erythromycin and fumagillin now under trial may prove to be equally effective against both hepatic and intestinal amebiasis.
- e General supportive measures should be instituted as for infectious hepatitis (see p. 279). A 2 week rest period may be followed by a repeat course of treatment. After the patient has convalesced from his hepatic disease further anti amebic drug therapy might be considered in an effort to eradicate the intestinal infection.

### 2 Measures for liver abscess

- a Treat as for hepatitis (see above). If patient responds to chloroquine or emetine treatment follow up with other amebicides for a period of 2 weeks (see p. 480). A repeat course alternating these drugs may be necessary after a rest period of one week.
- b Small abscess. If patient responds to hepatitis treatment use combined and then alternating courses of chloroquine or emetine and other drug therapy (see above).
- c Large abscess. If patient does not show definite response to emetine or chloroquine treatment.
  - (1) Continue treatment and carefully attempt to localize abscess site by physical and x ray findings. Aspirate under aseptic conditions (preferably operating room) and repeat aspiration if necessary. Avoid open drainage unless abscess is secondarily infected.
  - (2) Continue drug therapy (see above) until evidence of both hepatic and intestinal disease is eradicated.
- d Secondarily infected abscess.
  - (1) If aspirated material reveals pus and organisms (by smear and culture) it may be necessary to establish open drainage (by extra serous approaches).
  - (2) Chemotherapeutic agents should be employed in these cases (see pp. 496-514).
  - (3) Complete course of anti amebic therapy as for hepatitis or liver abscess if indicated (see above).

### 3 Observe for toxic reactions (see p. 481)

## D Amebiasis of Other Organs. Specific therapy as for acute or chronic amebic dysentery.

**P Asymptomatic Amebiasis (Carrier State)**

1 Follow-up Some clinicians feel that amebiasis always has systemic as well as local effects; they therefore recommend a full course of therapy for acute or chronic dysentery (see p 480).

2 Simple oral ambulatory treatment is considered satisfactory by other clinicians:

- a Carbarsone 0.33 Gm (3 $\frac{1}{2}$  gr) t i d orally for 7 days
- b Vioform® 0.33 Gm (3 $\frac{1}{2}$  gr) t i d orally for 10 days
- c Follow-up stool examinations should be performed as for active amebic infection (see p 481).

Follow-up Patients should be followed by clinical and laboratory examinations on at least one occasion each month for 3 to 6 months following therapy. Examination should include sigmoidoscopy and study of fresh stools on 3 successive days (preferably at least 1 following saline catharsis). Repeat examinations should be performed within a year if necessary. Need for chemotherapy must be based upon actual demonstration of amebiasis rather than mere presence of symptoms (e.g. chronic diarrhea). Consider the possibility of complications of the disease as secondary infection, irritation of bowel from chemotherapy, psychic trauma, etc. when persistent symptoms are not substantiated by laboratory findings. Avoid over-treatment with emetine or other drugs.

**GIARDIASIS (code No 604.155)**

Giardiasis is manifested by recurrent episodes of diarrhea consisting of watery or semi-solid bulky and often foul-smelling stools. A mild epigastric pain may occur. The specific diagnosis is made by demonstration of *Giardia lamblia* (tritypanis) in the stools.

**Treatment**

- A Quinacrine Hydrochloride U.S.P. Mepacrin Hydrochloride B.P. (Aureo-lin®) 0.1 Gm (1½ gr) t i d for 3 days
- B Repeat stool examination after one week to determine efficacy of treatment. Repeat treatment if necessary.

**DISEASES DUE TO METAZOA****TRICHINOSIS (code No 255)**

Trichinosis is caused by the ingestion of raw or undercooked infected pork. It may also be traced to other host animals which have consumed the same infected food. The incubation period is from 2 days to 4 weeks.

- A Antiparasitic treatment may be very mild or may be fatal. The gastroenteric symptoms occur early and are followed by fever 1 to 3 days by eye pain and other involvement.
- 1 Control the final symptom. It may vary from simple diarrhea to fatal convulsions.

- 2 Constitutional symptoms Fever chilliness weakness
- 3 Skin Rash periorbital and dependent edema splinter hemorrhages
- 4 Muscles Pain and tenderness in muscles
- 5 C N S May show any central or peripheral neurological involvement headache prominent
- 6 Eosinophilia
- 7 Trichinella skin test Delayed reaction (noted only after 12 to 36 hours) Occurs early in the disease (3rd to 7th day)

#### B Chronic Manifestations

- 1 Vague symptoms Weakness and other symptoms referable to multiple organ systems
- 2 Eosinophilia Often marked
- 3 Trichinella skin test Immediate reaction (noted after 5 minutes) occurs late in the disease (from 17th day on)
- 4 Muscle biopsy may demonstrate organisms

#### Treatment.

- A Supportive and Symptomatic Severe acute cases require hospitalization and excellent nursing care
- B No effective specific therapeutic agent is available
- C Corticotropin (ACTH) and cortisone or hydrocortisone provide effective relief for the acute symptoms of trichinosis. A reduction of the eosinophil count disappearance of fever and splinter hemorrhages if present and a general improvement in the clinical state of the patient are guides which should be employed to determine the efficacy of treatment
  - 1 Acute stage Treat with relatively large doses of either drug for first 24 to 48 hours (see p 423)
  - 2 Subacute stage Therapy may have to be continued for several days or weeks to prevent recurrence of symptoms. The dosage is reduced keeping symptoms under control (see p 423)

### ASCARIASIS OR ROUNDWORM DISEASE (code No 650 241)

Infection with *Ascaris lumbricoides* may cause no symptoms or only ill-defined gastrointestinal and nervous symptoms. Occasionally generalized urticarial reaction may develop rarely ascariasis pneumonia may result. The specific diagnosis is made by finding the worm's eggs or adult worms in stools or vomitus or by observation of the adult worm passing from nose or mouth.

#### Treatment.

- A Hexylresorcinol U S P (drug of choice for adults)
  - 1 Initial purgation Give 30 Gm (1 oz) magnesium sulfate in water or 240 cc (8 oz) of solution of magnesium citrate the night before drug therapy
  - 2 A light meal is given the preceding evening and then no further food until at least 5 hours after taking the hexylresorcinol
  - 3 Alcohol is contraindicated before and during treatment
  - 4 Hexylresorcinol 5 hard gelatin capsules 0.2 Gm (3 gr)

(crystoids) (total 1.0 Gm. 15 gr.) are given in the morning on an empty stomach. These are to be swallowed whole not chewed. Doses for children: Under 5 yrs. of age 0.4 Gm. (6 gr.) 5 to 8 yrs. 0.6 Gm. (9 gr.) 8 to 12 years 0.8 Gm. (12 gr.)

5 Purgation. Two hours later give 30 Gm. (1 oz.) magnesium sulfate in water to remove the worms from the bowel. Repeat 12 hours later if necessary for purgation.

6 Stool examination should be made once or twice later on 3 successive days to determine efficacy of treatment.

7 Treatment may be repeated in 3 days if necessary.

B Dithyrc + thiamine (H. Traub). Method of choice for the treatment of anemia in children. Give 12 mg. per Kg. body weight once a day for 4 days. Pre-treatment fasting and post-treatment purgation are not necessary.

C Oil of Chenopodium + Trichloroethylene. May be used if hexylresorcinol is unavailable. Caution. Trichloroethylene stimulates activity of esophagus and may result in bowel obstruction. A preliminary course of hexylresorcinol is advised before using the combined method.

1 Follow procedure of treatment as mentioned above for hexylresorcinol.

2 Oil of chenopodium 0.3 cc (4½m) apoc. and trichloroethylene 3.0 cc (45m) soluble gelatin capsules (total dose 3.3 cc 45m) are given together and followed by purgation as mentioned above.

## ANCYLOSTOMIASIS OR BOOKWORM DISEASE

(code No. 450-243)

Most commonly caused by *N. catenaticus* (New World) or *Ancylostoma duodenale*. The diseases are characterized by the number of the worms are present in the intestine. It is manifested by fatigue, weakness, dyspnea, palpitation, anorexia, inverted appetite, weight loss and a mild microcytic anemia. Ground itch, an erythematous or macropapular or vesicular pruritic dermatitis may develop at the site of penetration of the skin by the larvae. The specific diagnosis is made by finding the characteristic eggs in the stool.

Recent work indicates that symptoms are most often due to a coexisting deficiency disease. Correction of the malnutrition by adequate therapy as well as of the anemia by the addition of iron appears to alleviate or remove the manifestations in the absence of specific treatment for the bookworm infection.

### TREATMENT

A General Therapy. Estimation of the need for treatment should be based upon quantitative counts of the eggs in the stools. There is no indication for treating light infections, particularly after completion of previous therapy. It is often possible to completely eradicate the infection.

1 Correct malnutrition. Provide an adequate protein diet with supplemental iron medication (see p. 118).

2 Reduce possibility of reinfection: a. If

- 3 Gentian Violet U S P    Crystal Violet B P (4 hour enteric coated tablets) 1 mg per lb body weight divided into 3 daily doses a c Administer daily for 8 days follow with a rest period of 8 days and then administer again for another 8 days

**TAPEWORM INFECTIONS (code No 604 261)**  
(Pork Beef Fish Dwarf or more rarely Dog or Rat Tapeworms)

Diagnosis

- A History of consumption of raw or incompletely cooked pork beef fresh water fish or other contaminated food  
B Acute Phase (usually after a long incubation period of 3 to 4 months) Diarrhea fever leukocytosis and eosinophilia  
C Chronic Phase Vague gastrointestinal and C N S symptoms mild to severe anemia and presence of gravid proglottids in feces or on underclothing  
D Specific Diagnosis

Species and Source	Best Demonstrated By
<i>Taenia saginata</i> (beef)	Gravid proglottids in feces (occasionally by eggs in feces)
<i>Taenia solium</i> (pork)	
<i>Dipylidium caninum</i> (dog)	
<i>Diphyllobothrium latum</i> (fish)	Eggs in feces
<i>Hymenolepis nana</i> (dwarf)	
<i>Hymenolepis diminuta</i> (rat)	

Treatment

A Specific Measures

- 1 Quinacrine Hydrochloride U S P    Mepacrine Hydrochloride B M (Atabrine®)  
a On day preceding treatment patient eats a light low residue lunch and supper  
b Sodium sulfate or magnesium sulfate 15 to 30 Gm (½ 1 oz) cathartic the night before treatment  
c On morning of treatment omit breakfast Give pheno barbital 30 to 60 mg (½ to 1½ gr) depending upon the weight and age of the patient One hour later administer two 0.1 Gm (1½ gr) tablets of quinacrine hydrochloride every 5 min tes with a glass of water containing ½ tea spoon of sodium bicarbonate until 8 tablets have been given  
d Sodium sulfate or magnesium sulfate 15 to 30 Gm (½ 1 oz) in water is given two hours following therapy to rid the intestine of the parasite No food should be permitted until the bowels move copiously  
e The patient should have his evacuations in a basin of warm water to facilitate procurement and identification of the head and proglottids Toilet paper should be disposed of separately Examine all stools passed during the next 24 hours in order to recover the worm head for proof of complete eradication  
f Repeat course of treatment after 7 days if necessary

## 2. Hexylresorcinol U.S.P.

a. 10 Gm (15 gr) dissolved in 20 cc water introduced into the duodenum by a tube. Follow in two hours with sodium sulf to a magnesium sulfate. Examine stools for head of worm.

b. Crystoids antihelminthic as administered in ascariasis (p. 484) is the drug of choice for the treatment of Hymenol pliantha (dwarf tapeworm) infections.

3. Aspidium Oleraceum U.S.P. Extract of Male Fern B.P. a. Contraindications: Severe cardiac, hepatic or renal disease, constipation, acute or chronic gastroenteritis, febrile states, pregnancy and infancy.

b. Semi starvation, fat free diet for 24 to 48 hours prior to drug therapy. Lunch and supper should be omitted on the day before treatment. Alcohol is contraindicated.

c. Magnesium sulfate or sodium sulfate 15 to 30 Gm (1/2 lb) in water is given at 6-00 p.m. the night before and again in the morning before administering the drug.

d. A pipidimol orcinol  
A. a. la  
Distilled water q.s.d. 600 gr  
Give half this solution at about 7:00 a.m. orally or by duodenal tube and an hour later give the remaining half.

e. Magnesium sulfate or sodium sulfate 15 to 30 Gm (1/2 lb) in water is given again 2 to 3 hours after the second dose of aspidium in order to rid the intestine of the parasite as well as the drug. Give a soap suds enema 2 hours after the second cathartic. Food should be permitted until the bowels move copiously.  
Repeat course of treatment in not less than 7 days if necessary.

## SYSTEMIC MYCOSES

Myotic infections are caused by variety of fungi and have a wide geographical distribution. Although their incidence is rather low, a great part of the world seems of them occur quite commonly. *Coccidioidomycosis* in the San Joaquin Valley of California. The incidence in all nations is exceedingly variable with some resemblance to the granulomatous diseases.

### COCCIDIOIDOMYCOSIS (code No. 012 319) (Pulmonary code No. 360 219)

Coccidioidomycosis or Valley Fever is an infection due to *Coccidioides immitis* which is found in the Southwest United States, Mexico and Central and South America with periodic epidemics in Italy and Hawaii.

**Form I**  
a. Cures the bronchi and lungs and may mend rough erythema nodosum and purpura of the lungs during the primary disease.  
b. X-ray

shows patchy soft infiltration as this clears residual nodular shadows may persist. Thin walled cavities with little surrounding infiltration may develop and remain for months. The sedimentation rate is elevated. Organisms may be found in the sputum by culture and the skin test may be positive after 10 to 14 days. Complement fixation and precipitin tests are helpful in establishing a diagnosis and may aid in determining the progress of infection.

- II Chronic or Granulomatous Form.** 0.2 per cent of all primary cases progress to the granulomatous stage involving the lungs, chest wall or other structures. In the granulomatous stage the finding of the organisms in infected tissue or in the discharge from the lesions makes the diagnosis. Prognosis in this form is poor.

### Treatment.

No specific therapy is known for either form of the disease.

- A Primary Form.** Bed rest and symptomatic care until process has subsided.
- B Chronic Form.** Treatment entirely symptomatic. Potassium iodide is of no value and may even be dangerous.

**ACTINOMYCOSIS (Regional code No 0 202)**

**NOCARDIOSIS (Pulmonary code No 360 201)**

Actinomycosis is world wide in distribution and is caused by an anaerobic actinomycete *Actinomyces bovis*. Nocardiosis is caused by a variety of aerobic types belonging to the genus *Nocardia* (e.g. *N. asteroides*, *N. madrae*).

### Diagnosis

- A Actinomycosis.** The principal lesions are multiple abscesses, sinuses and fistulous tracts which discharge a sanguino-purulent material containing sulfur granules. Any region of the body may be infected but the head and neck are most frequently involved in which case very little systemic reaction occurs. The abdominal viscera may be involved by way of the intestinal tract or the lungs, pleura and chest by way of the respiratory tract. In the latter two forms there may be symptoms referable to the system affected accompanied by chills and fever. The finding of the typical sulfur granule in the lesion is diagnostic.
- B Nocardia infections** may resemble classical actinomycosis with the production of characteristic granules or produce a pseudotuberculous involvement of the lungs and pleura with extension at times to brain and meninges. Various species of *Nocardia* cause infections of the subcutaneous tissues with bone involvement (mycetoma).

### Treatment.

- A Actinomycosis.** The treatment of actinomycosis must frequently be continued for weeks or months.

1. Penicillin is the drug of choice. An initial dose of 120,000 units should be followed by 80,000 units every 4 hours. The

use of much higher dose is recommended by some clinicians daily intramuscular injections of 600,000 units for systemic fungal infections and one to two million units per day for the pulmonary and bronchial types.

2. Sulfadiazine The concomitant use of sulfadiazine has been found beneficial in certain instances.

3. Potassium Iodide With some patients the above combined medication should be supplemented by iodide therapy but extreme caution must be used in sensitive patients. Give saturated solution of Potassium Iodide U.S.P. 3 drops 4 times daily increasing each dose by one drop 4 times daily until reactions occur or a total of 25 drops 4 times daily is reached. This may be further increased and as much as 100 drops 4 times daily may be used if the patient tolerates this dosage. If reactions occur stop drug and begin again at a lower dosage. In a sensitive patient it may be necessary to start with 1 or 2 drops daily and gradually increase dose.

B. Non-fungal Treat as for actinomycosis. The diseases producing serbriacosis are more susceptible to the sulfonamides than is *A. bovis*. Treat with a fluid slow release or combine with a sulfamerazine. Maintain blood level of 10 to 20 mg per 100 cc until infection is controlled. Patient should be maintained on sulfonamide therapy 3 to 6 months following appearance of cure.

### BLASTOMYCOSIS

(Generalized code No 012.300) (Of Skin code No 210.217)

North American blastomycosis chiefly is found in the Carolina, Florida and Mississippi due to *Blastomyces dermatitidis*. The South American variety is due to *B. brasiliensis* and is found mainly in Brazil.

#### Diagnosis

The North American variety produces granulomatous lesions of the skin and subcutaneous tissue of the lungs and at times of other structures. The chronic form usually is without systemic symptoms but the pulmonary form usually has symptoms similar to those of pulmonary tuberculosis.

The South American variety involves the mucous membranes of the mouth, the skin, the lymphatic system and the viscera.

The diagnosis of blastomycosis depends primarily on recognition of the characteristic large thick-walled fungus in tissue smears followed by positive results of the organism's culture.

#### Treatment

A Potassium Iodide therapy should be given for blastomycosis (see above). Before administering this drug the patient should be tested sensitively to the drug and to iodine. If these reactions are not observed give 2 cc of the saturated potassium iodide solution 4 times daily. At the beginning of the treatment a small amount of potassium iodide should be used if there is danger of precipitation of the iodine on dermatitis or other lesions. Continue the therapy until the lesions are cured.



- B Stilbamidine has proved to be quite effective in the treatment of cutaneous and systemic blastomycosis. Dose is 10 to 200 mg daily not exceeding 2 to 3 mg /Kg daily. It is given slowly I V in 5% glucose in water or in saline. A course of 30 days may be required. This drug has to be employed with caution since it is toxic and frequently produces a neuropathy especially of the fifth nerve. A related drug 2 hydroxystilbamidine has been used successfully on patients infected with *H. dermatidis*. It fortunately does not produce peripheral neuropathy.
- C X ray therapy may be used as an adjunct in the cutaneous cases. The systemic forms are rather resistant to treatment and progress in spite of therapy.

### HISTOPLASMOSIS (code No 610 218)

Histoplasmosis is caused by *Histoplasma capsulatum* a small yeast like organism in tissue and a mold like fungus in culture. It has a world wide distribution. It primarily involves the reticulo endothelial system causing enlargement of the liver spleen and lymph nodes and systemic manifestations of fever anemia and leukopenia. However other systems may be involved. Patients from endemic areas often have pulmonary calcifications negative tuberculin tests and positive histoplasma skin tests.

#### Treatment.

None known

### MONILIASIS

(Pulmonary code No 360 208)

(Thrush of Mouth code No 610 209)

Moniliasis is an infection caused by *Candida albicans* which usually affects the mucous membranes of the mouth and vagina and the skin and nails. It may rarely involve the lungs and meninges. The diagnosis of the pulmonary form may be difficult. Cough and scanty sputum are most common findings. X ray appearance is similar to that of tuberculosis but tubercle bacilli cannot be demonstrated in the sputum. One must demonstrate the constant presence of *Candida albicans* before the diagnosis can be entertained. However the organism may occur as a normal habitant of the throat so great care must be taken in making the diagnosis of pulmonary moniliasis.

#### Treatment.

- A Oral Infection. Alkaline mouth washes. Topical application of gentian violet diluted 1 in 600 in 10 per cent alcohol for 4 to 5 days.
- B Vaginal Infection. Alkaline douches douches of potassium permanganate 1:1500 or gentian violet 1 in 600 propionate vaginal jelly.
- C Cutaneous Infections. Soak involved parts twice daily in 1:2000 potassium permanganate for 30 minutes. Follow with 1 per cent gentian violet paint in 15 per cent alcohol.

## CRYPTOCOCCOSIS OR TORULOSIS

(Of Skin code No 110 21x) (Meningitis code No 910 21x)

Cryptococcosis involve chiefly the skin and central nervous system but may invade other structures. It is world wide in distribution and is caused by *Cryptococcus neoformans* (*Torula histolytica*). The cutaneous lesions are papules, granulomatous ulcers or nodules. Meningeal involvement is the usual central nervous system lesion. The disease is usually mistaken for tuberculous meningitis if the organisms are not found.

Treatment

None other than symptomatic.

## IMMUNIZATION SCHEDULES

Biologicals for immunization purposes are gradually being modified and concentrated. The schedules below do not apply to all preparations; follow the manufacturer's instructions which accompany the preparation.

Children

## 1 During first year

- a Uncombined method. Pertussis vaccine (20 billion organisms per cc) subcut injections of 1 cc, 2 cc and 2 cc at one month intervals beginning at 2 to 3 months of age. Diphtheria tetanus toxoid (alum or minimum by droside) subcut injections of 0.5, 1.0 and then 1.0 cc at one month intervals beginning at 6 months of age and malipox vaccination at 6 months to 1 year of age. Repeat if a rash does not occur.

- or b Combined method. Diphtheria pertussis tetanus (combined) subcut injections of 0.5, 1.0 and then 1.0 cc at one month intervals starting at 2 to 6 months of age and malipox vaccination as with uncombined method.

- 2 At two years. Schick test and booster dose of diphtheria pertussis tetanus mixture 1.0 cc by subcut injection.

- 3 At school age. Repeat procedure as for two years and do vaccination (repeat if a rash does not occur).

Adults

Adults traveling to foreign countries should obtain a list of required immunizations when applying for passports. Those living in endemic areas should maintain their immunization.

- 1 Smallpox vaccination. Repeat every 5 years or on exposure.
- 2 Typhoid (or typhoid paratyphoid) vaccine (1 billion organisms per cc) 0.5, 1.0 and then 1.0 cc by subcut injection at weekly intervals. Repeat every 3 years.
- 3 Yellow fever vaccine (Africa South America) 0.5 cc subcut.
- 4 Typhus vaccine (C type) (Europe Africa Australia South America and Mexico) 1.0 cc subcut repeat 4 to 10 days later for total of 2 doses. Repeat 1.0 cc every 4 to 6 months.
- 5 Cholera vaccine (Asia Near East East Indies) 0.5 and then 1.0 cc every 6 to 8 months.

- 6 Plague vaccine (2 billion organisms per cc) (Egypt Asia East Indies) 0.5 and 1.0 cc subcut at interval of 7 to 10 days
- 7 Tetanus toxoid 1.0 cc subcut repeated at 30 and 60 days for total of 3 doses. Booster injection of 1.0 cc 1 year later and on injury
- 8 Diphtheria immunization in adults who are Schick positive is frequently followed by severe local and general reactions. A skin test (0.1 cc of 1:20 dilution of fluid toxoid) should be applied intradermally. If negative 0.5, 1.0 and then 1.0 cc subcut may be given at monthly intervals. If positive inject intradermally 0.1 cc of 1:10 dilution of toxoid at 3 to 4 week intervals for 3 doses.

## SENSITIVITY TESTS AND DESENSITIZATION

### Sensitivity Tests

Before injecting antitoxin or similar other material derived from animal sources always perform the following two tests for sensitivity

- A Intradermal Test. Inject 0.1 cc of a 1:10 dilution of the antitoxin intradermally into the skin of the flexor surface of the forearm. A positive test is manifested within 30 minutes by the occurrence of a large wheal and surrounding areola.
- B Conjunctival Test. Instill 1 drop of a 1:10 dilution of the antitoxin into the conjunctival sac of one eye as a test dose and 1 drop of physiological saline in the other eye as a control. A positive test is indicated by conjunctival injection, itching and edema occurring within 30 minutes.

### Interpretation of Sensitivity Tests

- A Negative Test. If both tests are negative no desensitization is necessary and a full dose of the antitoxin may be given.
- B Positive Test. If one or both of the tests are positive desensitization is necessary. Proceed as below.

### Desensitization

#### A. Precautionary Measures

- 1 Antihistaminic drug should be administered before beginning desensitization in order to lessen any reaction that might occur (see p. 86).
- 2 Epinephrine U.S.P. Adrenaline H.P. 0.5 to 1.0 cc (8.15%) of a 1:1000 solution must be ready in a syringe for immediate administration.

- B Desensitization Method. The following plan may be used in desensitization. Give doses I.M. at 30 minute intervals and observe closely for reactions.

1st dose	0.1 cc (1:10 dilution)	7th dose	1.0 cc (undiluted)
2nd dose	0.2 cc (1:10 dilution)	8th and subsequent doses	
3rd dose	0.5 cc (1:10 dilution)	1.0 cc (undiluted) every	
4th dose	0.1 cc (undiluted)	30 minutes until the total	
5th dose	0.2 cc (undiluted)	amount of antitoxin is given	
6th dose	0.5 cc (undiluted)		

Treatment of Reactions

- A If mild reaction occurs drop back to the next low  $\equiv$  dose and continue with the desensitization. If severe reaction occurs administer epinephrine as mentioned below and discontinue the antitoxin unless this treatment is urgently needed. Should desensitization be imperative administer slowly using more gradual increase of the antitoxin.
- B If manifestations of a severe reaction appear give 0.5 to 1.0 cc (8-15 M) of epinephrine subcutaneous at once. The symptoms include urticaria, angioneurotic edema, dyspnea, coughing, choking or shock. Maintain close observation of the patient and repeat epinephrine as necessary.

## CHEMOTHERAPEUTIC AGENTS

The sulfonamide drugs and the antibiotics penicillin streptomycin chlortetracycline (Aureomycin®) chloramphenicol (Chloromycetin®) oxytetracycline (Terramycin®) tetracycline bacitracin polymyxin neomycin and erythromycin are powerful agents affecting a wide variety of pathogens. Each however has a definite and limited antimicrobial spectrum and beneficial effects can be obtained only in infections due to those organisms included in the spectrum. *Etiological diagnosis in infections is of paramount importance.*

Indiscriminate use of antibacterial agents is wasteful may lead to a false sense of security by doing something and may cause serious toxic effects. The sulfonamide drugs in particular are potentially dangerous agents and should be reserved for certain specific functions.

Certain chemotherapeutic agents e. g. penicillin and streptomycin may exert synergistic activity in infections notably those due to *Streptococcus fecalis*. On the other hand it has been shown in the laboratory that penicillin is antagonized by chloramphenicol chlortetracycline and oxytetracycline when the infecting organism is penicillin sensitive. The clinical importance of this antagonism is not known. At any rate the use of two or more antibiotics in combination is probably best avoided except under careful laboratory control.

### Indications for Use of Chemotherapeutic Agents

- 1 Against an infection due to a proved pathogen known to be susceptible to the proposed drug (e. g. subacute bacterial endocarditis due to *Streptococcus viridans* which is susceptible to penicillin)
- 2 Against an infection wherein a susceptible etiological agent may be reasonably assumed from the clinical picture to be present (e. g. lobar pneumonia due to a *Pneumococcus*)
- 3 As an attempted lifesaving measure against infections of obscure etiology. This category should be sharply limited
- 4 As prophylaxis against potential invaders (e. g. during and following tooth extraction in patients with valvular heart disease to prevent subacute bacterial endocarditis)

## SULFONAMIDE DRUGS

The sulfonamide drugs are a batch of derivatives of sulfanilamide. Newer derivatives have wider antibacterial spectra and more desirable pharmacological properties than the older sulfonamides. Since the activity of any sulfonamide compound may be predicted on the basis of certain physicochemical principles, it is evident that maximal antibacterial effectiveness has been approximated by sulfamazine, sulfamerazine, sulfamethazine and sulfisoxazole (Ganturin®) and the use of older sulfonamides is rarely if ever warranted.

### 1. Indications and Antibacterial Spectrum (See table on p. 514)

The sulfonamide drugs have a wide but still limited range of activity against pathogenic agents. At the present time the sulfonamides are the therapeutic agents of choice only in meningococcal infections (Nisseria meningitidis).

- A. Except for the infection mentioned above, the sulfonamides should be used as alternate or additional agents to one of the antibiotics against infections of known susceptibility.
- B. Promizol® Promizole® and Diasone® are members of the sulfonamide group which show promise within a limited area. They are usually used in addition to other agents against the following pathogens:
  1. Mycobacterium tuberculosis
  2. Mycobacterium leprae

### Absorption

The sulfonamides are poorly absorbed from the stomach but are readily absorbed from the small and large intestines. Peak blood concentrations are reached in from 2 (sulfanilamide) to 8 hours (sulfamerazine) following a single oral dose. The sodium salts are rapidly absorbed from intramuscular or subcutaneous sites.

### Distribution

- A. Distribution Through Body and into Body Fluids. The sulfonamides are rapidly distributed throughout the body and readily found in every tissue. They transmute saliva, gastric and intestinal secretions, pancreatic juice, milk, cervical and prostatic secretions, amniotic fluid and fetal blood. Sulfamerazine and sulfadiazine enter the cerebrospinal fluid and cerebrospinal fluid is approximately one-half the plasma concentration.
- B. A Little of Plasma Protein Binding and Excretion. Approximately one-third of the sulfonamides in the blood is bound to plasma proteins and is inactive. About 10 to 20 per cent is excreted by the liver and also is therapeutically inactive.

### Excretion

- A. Excretion in the Renal Region. The sulfonamides are almost entirely excreted by the kidney. The rate of excretion depends on the rate of urine flow rather than the plasma level. Tubular reabsorption of 10 to 20 per cent occurs. Impaired renal function may result in dangerous high blood concentrations and may be anticipated by means of the phenolphthalein

(P S P) test Sulfanilamide is excreted most rapidly and sulfamerazine most slowly necessitating less frequent administration

- B Solubility Factors in Excretion** The sulfonamides and their acetyl derivatives are more soluble in alkaline than in acid urine hence alkalization together with increasing urine output prevents precipitation of the drugs in the urinary tract Since each sulfonamide is soluble in the urine independently of the presence of any other the simultaneous administration of fractional doses of two or more sulfonamides further lessens the likelihood of precipitation while still permitting an effective blood level of combined sulfonamides Sulfisoxazole (Gantisin®) is highly soluble in the urine

### Mode of Action

- A Bacteriostatic Activity (Not Bactericidal)** The sulfonamides are bacteriostatic and depend on body defense mechanisms for final destruction of the pathogenic agents
- B Bacteriostasis Occurs by Interference with Growth and Division** The sulfonamides act against bacteria by reason of their structural similarity to para aminobenzoic acid (PABA) which is an essential substrate for certain necessary enzyme activities of bacteria The sulfonamide molecule when in excess of para aminobenzoic acid is substituted for the natural substrate of the enzyme system and thus renders the organism incapable of growth and multiplication
- C Natural and Acquired Resistance to Sulfonamide** Bacteria may be naturally resistant to the action of sulfonamides by reason of being able to synthesize para aminobenzoic acid (PABA) or may acquire resistance when allowed to exist in a low concentration of sulfonamide In this instance only individual organisms best able to synthesize PABA survive and multiply
- D Neutralizing Effect of Para aminobenzoic Acid (PABA) in Sulfonamide Therapy** PABA is a fraction of vitamin B complex and will compete with the sulfonamides in the enzyme activity of the bacteria and so interfere with the antibacterial effects of the sulfonamides Large amounts of crude B complex or liver extract should not be given to patients under sulfonamide therapy Procaine and certain other local anesthetics are esters of PABA and may exert local anti sulfonamide activity PABA should be included in culture media used to isolate organisms from patients receiving sulfonamides so that the drug present in the body fluid which is cultured may not alter the true bacteriological picture Exudates and other substances which promote bacterial growth inhibit sulfonamide activity

### Blood Levels

Under most circumstances effective blood levels will be attained by following standard dosage recommendations Insufficient blood levels may be followed by development of sulfonamide resistance by the infecting organism Since urine concentrations are 10 to 20 times that of the blood the dosage in urinary tract infections unaccompanied by marked tissue invasion or bacteremia may be

- A Blood Levels of Sulfonamide Should Be Determined Under the Following Circumstances**

- 1 Repeated parenteral administration
- 2 Lack of expected therapeutic effect
- 3 Unusually high doses
- 4 If renal insufficiency is a suspected known

#### B Optimal Levels (Blood)

Sulf dia 0.4-15 mg %	Sulfanilamide 8-15 mg %
Sulfamazine 8-15 mg %	Sulf pyridine 3-10 mg %
Sulf thiazole 3-7 mg %	

#### Dose and Methods of Administration

##### A Oral

##### 1 Adults

##### a Most infections

- (1) Initial dose 2 to 4 Gm (30-60 g) of one of the sulfonamides or of a mixture of sulfonamides
- (2) Maintenance dose

(1) 0.5 Gm (7.5 g) 4 or 6 times a day (if possible, use the preferred method)

or (b) 1.0 Gm (15 g) of sulfonamide (Gm 1.0) every 6 hours

or (c) 0.33 Gm (5 gr) each of sulfamazine and sulfathiazole every 4 to 6 hours

or (d) 1 Gm (15 g) of sulfanilamide if pyridine is thiophene every 4 hours

or (e) 1 Gm (15 gr) of sulfadiazine every 4 to 6 hours

or (f) 1 Gm (15 gr) of sulfamazine every 6 to 8 hours

b Urinary tract infections 0.5 to 1.0 Gm (7.5-15 g) of one of the sulfonamides or of a mixture of sulfonamides every 4 to 6 hours

Prophylaxis of streptococcal infections 0.5 Gm (7.5 g) of one of the sulfonamides or of a mixture of sulfonamides twice daily

c Intestinal infection Initial dose of 0.03 Gm (3/4 gr) /lb body wt (1/2 Gm /Kg) of one of the sulfonamides or of a mixture of sulfonamides and follow up by 0.03 Gm (3/4 gr) /lb body wt (0.033 Gm /Kg) every 4 hours

d Leprosy and tuberculous (relapse) (1) Promin 4 c (15 g) daily I.V. for 3 weeks then rest one week and repeat a frequent

or (2) Promucol 1 to 2 Gm (15-30 g) daily by mouth divided dose

##### 2 Children

##### a Most infections

- (1) Initial dose 0.03 Gm (3/4 g) /lb body wt (0.045 Gm /Kg) of one of the sulfonamides or of a mixture of sulfonamides
- (2) Maintenance dose

(1) 0.03 Gm (3/4 gr) /lb body wt (0.045 Gm /Kg) 4 or 6 times a day (if possible, use the preferred method)

or (b) 0.01 Gm (1/4 gr) /lb body wt (0.01 Gm /Kg) of a mixture of sulfonamides every 4 to 6 hours

or (c) 0.03 Gm (3/4 gr) /lb body wt (0.03 Gm /Kg) every 4 to 6 hours

or (d) 0.03 Gm (3/4 gr) /lb body wt (0.03 Gm /Kg) every 4 to 6 hours

or (e) 0.03 Gm (3/4 gr) /lb body wt (0.03 Gm /Kg) every 4 to 6 hours



- or (d) 0.01 Gm ( $\frac{1}{4}$  gr) /lb body wt (0.02 Gm /Kg) sulfanilamide sulfapyridine or sulfathiazole every 4 hours
- or (e) 0.01 Gm ( $\frac{1}{4}$  gr) /lb body wt (0.02 Gm /Kg) of sulfadiazine every 4 to 8 hours
- or (f) 0.01 Gm ( $\frac{1}{4}$  gr) /lb body wt (0.02 Gm /Kg) of sulfamerazine every 8 to 8 hours
- b Urinary tract infections 0.003 to 0.01 Gm ( $\frac{1}{12}$   $\frac{1}{4}$  gr) /lb body wt (0.01 to 0.02 Gm /Kg) of any of the sulfonamides or of a mixture of sulfonamides every 4 to 8 hours
- c Prophylaxis of streptococcal infections 0.005 Gm ( $\frac{1}{12}$  gr) /lb body wt (0.01 Gm /Kg) of any of the sulfonamides or of a mixture of sulfonamides twice daily
- d Intestinal infections Initial dose of 0.03 Gm ( $\frac{3}{4}$  gr) /lb body wt (0.11 Gm /Kg) of sulfaguanidine or Sulfanixidine<sup>®</sup> followed by 0.03 Gm ( $\frac{3}{8}$  gr) /lb body wt (0.033 Gm /Kg) every 4 hours
- e Leprosy and tuberculosis (oral and I.V. doses)
  - (1) Promin<sup>®</sup> 0.3 to 0.6 cc (8 min  $\frac{3}{4}$  dr) daily I.V. for 3 weeks then rest one week and repeat course as frequently as needed
  - or (2) Fromsole<sup>®</sup> 0.25 to 0.6 Gm ( $\frac{3}{4}$  to 15 gr) every 8 hours orally

## II Intramuscular, Intravenous

### 1 Adults

- a Initial dose of 3 to 5 Gm (45 to 75 gr) of any sulfonamide (sodium salt) except sulfanilamide followed by 2 to 3 Gm (30 to 45 gr) every 6 to 12 hours (optimal interval to be determined by blood level just before second dose and occasionally thereafter)
- b Diluent may be physiological saline solution Ringer's solution  $\frac{1}{2}$  molar sodium lactate solution or Ringer's lactate solution
- c Concentration ideally approximately 0.5 per cent but may be as high as 5 per cent
- 2 Children As in adults Initial dose 0.03 to 0.05 Gm ( $\frac{1}{2}$   $\frac{3}{4}$  gr) /lb body wt (0.066 to 0.11 Gm /Kg) of any of the sulfonamides except sulfanilamide followed by 0.015 to 0.03 Gm ( $\frac{1}{4}$   $\frac{1}{2}$  gr) /lb body wt (0.033 to 0.066 Gm /Kg) every 6 to 12 hours

## Toxicity and Management

### A Toxic Reactions

- 1 Mild Continue therapy if necessary Symptoms include nausea vomiting headache dizziness crystalluria
- 2 Moderate Stop therapy unless continuation is essential to life Symptoms include fever rash stomatitis conjunctivitis arthritis diarrhea microhematuria psychosis
- 3 Severe Stop therapy and push fluids Symptoms include granulocytopenia hemolytic anemia aplastic anemia thrombocytopenia hepatitis exfoliative dermatitis severe hematuria oliguria leukemoid reaction

- II Allergic Reactions A considerable percentage of individuals who have previously received sulfonamides especially for more than 7 days become sensitized and may develop

Immediate and severe reactions on readministration. Fever, angioneurotic edema, urticarial and other rashes, and periarthritis nodosa may occur.

History of previous administration should be obtained. Cross sensitivity to various sulfonamides may exist. Severe symptoms may be avoided by giving a test dose of 0.5 Gm (7½ gr) and observing for 6 hours.

- Precautions**
- 1 Hemoglobin determination and white blood cell count every other day. Differential if WBC is less than 5000. Discontinue sulfonamides if granulocyte count is less than 50%.
  - 2 Daily fresh urine for pH (use nitrazine paper) and sediment. Increase alkali (sodium bicarbonate) if pH is less than 7.0. Discontinue drug if red blood cells are found in urine (see above). Increase urine output if less than 1500 cc per day or crystalluria occurs (must be examined for in a fresh specimen).
  - 3 Daily observation of patient for drug fever, rash, jaundice, nausea, vomiting, etc.

**Contraindications to Sulfonamides**

- 1 History of previous severe reaction.
- 2 Renal insufficiency (Very small doses may be used with caution).
- 3 Liver damage (Proceed with caution if essential).
- 4 Heart failure (If sulfonamides are absolutely necessary, substitute potassium bicarbonate for sodium bicarbonate and alkalinizing agent).

**PARA AMINO SALICYLIC ACID (PAS)**

Para amino salicylic acid (PAS) and its sodium salt have been found to exert considerable tuberculostatic activity. Tubercle bacilli resistant to streptomycin may be susceptible to PAS, and vice versa. The simultaneous administration of PAS and streptomycin delays the emergence of strains of tubercle bacilli resistant to the latter. In addition to the bacteriostatic effect, a diaphoretic activity is present. PAS is absorbed readily from the gastrointestinal tract. Peak serum concentrations are reached in 30 to 60 minutes and minimum levels are again reached in 6 hours. PAS may be administered intravenously and subcutaneously.

**Dosage**

- Adult** 2 to 3 Gm (30-45 gr) every 6 hours.  
**Prevention** 15 Gm in 3% solution given in 3 doses 6 hours apart.  
**Child** 5 mg of biparic should be added to each liter.

**Contraindications**

Nausea and vomiting, diarrhea, drug fever, dermatitis, crystaluria, hematuria, and hypoprothrombinemia may be observed. Gastrointestinal symptoms may apparently be avoided by parenteral administration of sodium PAS.

## ISONIAZID (INH)

Isoniazid (INH) and related compounds possess considerable tuberculostatic activity. Cross resistance to streptomycin and PAS does not exist. Bacterial resistance to INH develops rapidly. INH is readily absorbed from the gastrointestinal tract and distributed throughout the body fluids including the cerebrospinal fluid.

### Dosage

Isoniazid 3 to 5 mg (120 1/2 gr) /Kg body weight per day divided into 2 or 3 doses and given orally.

### Toxicity

Constipation, difficulty at micturition, increased reflexes, postural hypotension and dizziness, eosinophilia, slight anaemia, occasional casts and trace of albumin in the urine, reducing substances in the urine.

## PENICILLIN

Penicillin is prepared from the cultural products of the molds *Penicillium notatum* and *Penicillium chrysogenum*. The commercially available preparations are crystalline sodium, calcium, potassium, and procaine salts of penicilloic acid.

Four types of penicillin F, G, X and K are produced by the mold. Commercial penicillin is principally penicillin G. Penicillin X, which occurs only in small amounts, exhibits a slightly different range of antibacterial activity. Penicillin K becomes bound to serum protein and is relatively inactive therapeutically.

The Oxford and International units of penicillin are measured in comparison to the bacterial inhibitory power of a standard penicillin. Crystalline sodium penicillin contains approximately 1500 units per milligram. Dried crystalline penicillin retains its potency indefinitely but watery solutions may deteriorate especially when not refrigerated.

### Indications and Antibacterial Spectrum (See table p 514)

Penicillin exerts bacteriostatic and bactericidal activity against a wide variety of pathogenic agents but the susceptibility of these agents to penicillin may vary considerably. Clinical response of infections may be predicted with fair accuracy by means of in vitro sensitivity tests of the infecting organism. This procedure should be performed when expected therapeutic response does not occur or in the case of infections due to organisms such as staphylococci or *Streptococcus fecalis*, many strains of which are naturally resistant to penicillin.

Penicillin is indicated when infection with an organism known to be generally susceptible is diagnosed or presumed. Hence one treats a specific infection not a disease e.g. pneumococcal pneumonia, not pneumonia, streptococcal pharyngitis, not acute pharyngitis. For specific indications see under disease in question.

### Mode of Action, Resistance

Penicillin is both bacteriostatic and bactericidal. Its exact

mode of action is not known but in some way it apparently interferes with the reproductive process of organisms.

Certain organisms produce penicillinase which inhibits penicillin activity. This may occur naturally as in the case of *E. coli* and some strains of staphylococci or may be an acquired characteristic of once susceptible organisms existing in sublethal concentrations of penicillin. Those variants of the original organism which were naturally resistant survive and multiply while the susceptible organisms are destroyed. Acquired penicillin resistance is not commonly encountered clinically.

#### Aborption, Distribution, Excretion

A. Aborption Penicillin in watery solution is rapidly absorbed when administered intravenously or intramuscularly and somewhat more slowly absorbed after subcutaneous injection. The peak concentration in the blood is reached immediately after intravenous injection and within one hour after intramuscular injection. Blood levels persist for from 2 to 3 hours after doses of less than 500,000 units intramuscularly and somewhat longer with larger doses. Penicillin produces appreciable serum concentrations for 12 to 48 hours and for 96 hours when combined with 2% aluminum monostearate. Benzathine penicillin may produce measurable serum concentration for one month after injection of 600,000 to 1,200,000 units. With all repository forms maximum serum concentrations tend to be lower than with aqueous solutions and so are not appropriate where high serum concentration is desirable. Penicillin while not absorbed from the stomach is absorbed readily from the small intestine. Approximately 5 times the intramuscular dose must be given to produce comparable blood levels. Acids and buffers tend to decrease the destructive effect of gastric juices and absorption is best when the stomach is empty. Penicillin is poorly absorbed from the rectum and inconsistently absorbed from the vagina.

The concentration of penicillin in serum and other body fluids may be measured by various bioassay methods. Determination of blood levels and comparison with the in vitro sensitivity of the organism are useful in infections due to resistant organisms.

B. Distribution Penicillin is distributed throughout the body fluids but penetrates the joint, pleura, peritoneum, and subarachnoid space irregularly. Penetration is more likely to occur in inflammation and is. Penicillin G diethylaminoethyl salt hydrochloride (Lorpen) penetrates to higher concentrations than penicillin G in the lungs and sputum. Penicillin persists in the tissues for a considerable time after it has disappeared from the blood, hence continuous blood levels are not necessary in most infections. Organisms exposed to penicillin do not multiply for considerable time after exposure.

C. Excretion Penicillin is excreted principally in the urine 2% of a daily excretion is tubular and may be partly blocked by such agents as carbenicillin, para-aminosalicylic acid, Dacarbazine and Benemidol.

PreparationsA Commercially Available Preparations Include

- 1 Crystalline penicillin (sodium potassium salts)
- 2 Penicillin procaine in oil
- 3 Penicillin procaine in oil with 2% aluminum monostearate
- 4 Penicillin procaine in aqueous suspension (may be combined with crystalline penicillin sodium)
- 5 Penicillin tablets with or without buffers and bindings (30 000 to 200 000 units per tablet) and benzathcil penicillin suspension (60 000 units per cc ) for oral use
- 6 Penicillin powder for insufflation (50 000 units per cartridge)
- 7 Penicillin in ointments in various bases (general and ophthalmic) (300 2000 units per gram)
- 8 Penicillin G diethylaminoethyl ester hydroiodide (estopen Neo penit®) 300 000 units per cc

B Strength of Solutions and Suspensions

- 1 Crystalline penicillin is generally given in a concentration of 10 000 units per cc but may be much more concentrated
- 2 Penicillin for subarachnoid injection should not be more concentrated than 1000 units per cc
- 3 Penicillin procaine complex in aqueous suspension may be prepared in concentration of 300 000 1 200 000 units per cc

Dosage and Methods of Administration

- A Intermittent Intramuscular. Penicillin in aqueous solution may be given in doses of 5000 to several million units every 3 hours intramuscularly. This remains the method of choice in most severe acute infections. In many infections equally good results may be obtained by administration of 100 000 to 300 000 units every 12 hours intramuscularly. Intramuscular injection of 300 000 to 600 000 units of penicillin procaine may be given every 24 hours. Penicillin procaine in oil with 2% aluminum monostearate produces measurable blood levels which may persist as long as 96 hours. Benzathcil penicillin 600 000 to 1 200 000 units produces measurable serum concentrations for one month and is ideally suited to prophylactic use. These preparations are highly satisfactory except in the most severe acute infections.
- B Continuous Intramuscular and Continuous Intravenous. Where very high doses of penicillin are necessary in the treatment of infections due to resistant organisms administration by continuous drip is often advantageous. Many million units dissolved in 1000 to 2000 cc of physiological saline or 5% glucose solutions may be given by indwelling needle or catheter in 24 hours. The intramuscular site should be changed as frequently as irritation occurs. Thrombophlebitis as a complication of intravenous administration may be avoided by changing the vein used or by addition of 10 mg heparin sodium to the solution.
- C Oral. Penicillins may be given orally in all but the severest of infections or oral medication may be substituted for parenteral after initial response to treatment. Doses of 100 000 units every 3 hours to 300 000 units every 6 or 8 hours may be given.
- D Topical
- 1 Aerosol 50 000 to 100 000 units may be aerosolized from

3 to 8 times a day. A solution containing 50 000 units per 0.5 cc. may be nebulized by means of Vaponephrin® or DeVilbiss #40® nebulizers. Forced deep inhalation followed by retention of the inspired penicillin as long as possible should be insured. Hand pumping or compressed gas fed through a Y tube may be used to nebulize the solution. Penicillin powder in cartridges containing 50 000 units may be used similarly by means of the Abbott Aerosolator® or the Squibb Dispulator®. While local effect in the respiratory passages for the treatment of bronchiectasis, chronic bronchitis, etc., is usually the objective, appreciable blood concentrations of penicillin frequently result and natiua locution commonly.

2. Intrathecal. Although penicillin may penetrate the subarachnoid space after intramuscular injection, this phenomenon is inconstant and may be delayed. Therefore, in meningitis due to susceptible organisms, 10 000 units of penicillin dissolved in 10 cc. of physiological saline should be administered once a day until the cerebrospinal fluid glucose content becomes normal. Penicillin should also be given intramuscularly.
3. Intrapleural, intra-articular. 10 000 to 300 000 units of penicillin may be introduced into joint or pleural spaces infected by susceptible organisms daily or every other day following aspiration.
4. Oral. Troches of penicillin may be dissolved slowly in the mouth in the treatment of Vincent's stomatitis and pharyngitis. This form of therapy is valueless in other forms of pharyngitis and may produce stomatitis.
5. Wounds and skin. Solutions of penicillin containing 200 000 units per cc. may be used as a wet dressing in infected wounds. Penicillin is of no value as an irrigating solution because of the necessity of prolonged contact to produce antimicrobial effect.

Crystalline or penicillin incorporated in various bases may be used on infections of the skin due to susceptible organisms.

#### Toxicity

Since the purification of penicillin, true toxic reactions are almost unknown. Sensitization may be pre-existing or induced. Fever and rash, especially urticarial, may appear during the course of penicillin administration or as long as several weeks later. This may partly mimic serum sickness. True idiosyncrasy to penicillin is rare and may be largely limited to individuals suffering from dermatophytoses. Desensitization may be attempted. Patients known to be sensitive to penicillin may be treated with penicillin G or ethoxymethyl penicillin G, which may be substituted for aqueous and procaine penicillin G respectively in the same dose. Cross-sensitization occurs occasionally and should be guarded against.

## STREPTOMYCIN

Streptomycin is prepared from the cultural products of *Streptomyces griseus*. Commercially available salts include the sulfate hydrochloride and the calcium chloride complex. Dihydrostreptomycin may be used alternatively with streptomycin. Vestibular damage is less frequent following dihydrostreptomycin therapy but deafness occurs more often following prolonged dihydrostreptomycin therapy than streptomycin treatment. One microgram ( $\gamma$ ) equals 1 Wakeman unit and 1 Gm equals 1 000 000 Wakeman units.

Indications and Antibacterial Spectrum. (See table p 514.) Streptomycin is principally active against gram negative organisms but possesses significant activity against some strains of gram positive cocci. Penicillin and streptomycin exert marked synergistic activity in infections due to *Streptococcus fecalis* and streptomycin and Aureomycin® exert synergistic activity in brucellosis. Mycobacterium tuberculosis is highly susceptible to streptomycin.

The indications for streptomycin are almost entirely limited to infections due to gram negative organisms and tuberculosis. For this reason exact etiological diagnosis should be sought before instituting treatment. Streptomycin should be reserved for those cases of tuberculosis where a conservative rest regime would not be expected to produce good results. Combination with collapse therapy and surgical procedures often is of aid. Most tubercle bacilli become streptomycin resistant within 3 months of the beginning of treatment although the simultaneous use of PAS may delay this event.

### Mode of Action, Resistance

Streptomycin is both bacteriostatic and bactericidal. Its mode of action is unknown. Resistant variants of organisms may multiply quickly in infections treated with streptomycin so that further therapy with the antibiotic is useless. Streptomycin should be used only when necessary and at quite initial dosage should be used to prevent development of drug resistance.

### Absorption, Distribution, Excretion

- A Absorption.** Streptomycin is readily absorbed from the site of intramuscular injection. The peak serum concentration is reached within one hour and detectable amounts are present up to 8 hours later. It is likely that streptomycin persists longer than this in the tissues. If streptomycin is administered every 3 to 4 hours gradually increasing serum levels will be noted due to slow accumulation. Administration every 8 hours is sufficient in all but the most acute infections in which case the drug should be given initially every 3 or 4 hours. Streptomycin is not absorbed from the gastrointestinal tract but exerts bacteriostatic activity in the lumen of the bowel.
- B Distribution.** Streptomycin is distributed throughout the body similarly to penicillin. Penetration of the cerebrospinal fluid is inconsistent and unreliable.
- C Excretion.** Streptomycin is excreted principally in the urine where the concentration exceeds that in the serum.

# **Dosage and Method of Administration**

- A Intramuscular Injection** 1 to 5 Gm daily may be given in intramuscularly in divided doses very 3 to 6 hours. Most organisms realized infections require approximately 3 to 4 Gm per day. Urinary tract infections due to highly susceptible organisms may be treated with 250 mg intramuscularly every 6 hours for 3 to 5 days. Streptomycin should not be used in the presence of obstruction of the urinary tract because of the possibility of the development of resistant organisms.
- B Intravenous** In addition to intramuscular administration 25 to 30 mg daily in 10% of physiological saline solution may be given intravenously daily until the cerebrospinal fluid sugar content becomes normal.
- C Oral** Streptomycin may be administered orally 0.5 Gm (1/2 G) every 6 hours in the treatment of shigella dysenteriae.
- D Tuberculosis** 0.5 Gm of streptomycin and 0.3 Gm of dihydrostreptomycin intramuscularly twice weekly is indicated in the treatment of miliary tuberculosis. In the treatment of tuberculous pneumonia and miliary tuberculosis 40 mg/kg of body weight (20 mg/lb) daily should be given intravenously or intramuscularly in addition to 2 mg/kg of body weight (1 mg/lb) per day intrathecally (see Tuberculosis meningitis 80 mg/kg of body weight (40 mg/lb) daily should be administered intramuscularly in addition to 2 mg/kg of body weight (1 mg/lb) per day intrathecally (see Tuberculosis meningitis p 467).

## **Toxicity**

Infarct local reactions are uncommon. Systemic drug reactions of any sort are rare. Eosinophilia may be noted but appears to have no significance. Cytochrome and nitrogen retention are associated with permanent renal damage have been reported. In tubular damage of a manifest itself by tinnitus and characteristic severe vertigo. Adverse effects of high prolonged dosage of streptomycin include ototoxicity immediately follows streptomycin in the ototoxicity immediately follows streptomycin. Tubular damage may be permanent. Satisfactory compensation usually made by the patient. Deafness also may occur but dihydrostreptomycin but deafness may develop after treatment has been stopped. The use of combined streptomycin and dihydrostreptomycin in equimolar doses reduces the incidence of deafness and tubular damage. Pancytopenia is increased in patients with tubular damage. Pancytopenia is increased in patients with tubular damage. Pancytopenia is increased in patients with tubular damage.

## **CHLORTETRACYCLINE (AUREOMYCIN®)**

Chlortetracycline (Aureomycin®) is prepared from Streptomyces aureofaciens. It is available in the hydrochloride form. It is a broad spectrum antibiotic. It is a broad spectrum antibiotic. It is a broad spectrum antibiotic.



## 508 Oxytetracycline

spectrum antibiotic with a wide therapeutic range. It is active against most gram negative rods and gram positive cocci, the spirochetes of leptospirosis, relapsing fever, rat bite fever, syphilis and yaws, as well as the rickettsiae of typhus, Rocky Mountain spotted fever, scrub typhus, Q fever, and rickettsialpox. It is highly active against the viruses of psittacosis, lymphoplasma venereum, primary atypical pneumonia, and probably herpes zoster.

### Absorption Excretion

Chlortetracycline is absorbed slowly from the gastrointestinal tract and peak blood concentrations are reached in from 2 to 4 hours and persist as long as 12 to 24 hours depending on the dose. Intravenous administration produces an immediate high blood concentration which drops over a period of 6 to 24 hours varying with the dose. Chlortetracycline is poorly absorbed after muscular injection unless hyaluronidase is added. Accumulation occurs in the body at high dosage so that blood levels become increasingly elevated during prolonged administration at high dosage. Chlortetracycline is excreted slowly by the kidney. It does not appear readily in the cerebrospinal fluid or pleural fluid but it is present in high concentration in the urine and stools.

### Dosage

- A Oral.** 0.25 to 1.0 Gm. may be given orally every 6 hours in systemic infections and less frequently (every 8 to 10 hours) in urinary tract infections.
- B Intrav. or i.m.** Similar results may be obtained by the intravenous administration of 100 mg. every 6 to 8 hours or 500 mg. every 12 hours. In resistant infections combined oral and intravenous therapy may be used.
- C Intramuscular.** 250 mg. in 1% procaine solution with 250 units of hyaluronidase added every 6 hours may be substituted for intravenous therapy when required.

### Method of Administration

250 mg. orally every 6 hours appears adequate in most acute infections. Gastrointestinal symptoms may be minimized by administering the drug only when food is in the stomach or by simultaneously administering carboxymethylcellulose. Superinfections with yeast in the oropharynx and perineal area may occur but are probably secondary infections of local sensitivity reactions.

### Toxicity

Nausea and vomiting are common following oral administration but this may be avoided by reducing the dose to 250 mg. every 8 hours or administering the drug intravenously. Rashes and stomatitis may occur. Loose bowel movements may be observed.

## OXYTETRACYCLINE (TERRAMYCIN®)

Oxytetracycline (Terramycin®) is derived from *Streptomyces rimosus*. The commercial preparations are the hydrochloride and the base.

Indications and Anti Infective Spectrum (See table on p 314)  
 Oxytetracycline is a broad spectrum antibiotic whose range of activity is similar to that of chlorotetracycline. It may be used in infections due to gram positive and gram negative cocci, gram positive and gram negative rods, spirochetes, rickettsia and the viruses of primary atypical pneumonia, lymphoplasma virus and psittacosis.

#### Absorption and Excretion

Oxytetracycline is incompletely absorbed from the gastrointestinal tract. Salivary serum levels may be maintained by administration every 6 hours. Excretion is principally by the kidneys. Significant amounts appear in the bile. Appearance in the cerebrospinal fluid is delayed and irregular.

#### Dose and Route of Administration

- A Oral 0.25 to 1.0 Gm may be given orally every 6 hours.  
 B Intravenous 0.5 to 1.0 Gm dissolved in sodium glycinate buffer solution may be administered every 12 hours. Oral therapy should be used when possible.  
 C Intramuscular The preparation for I.M. use may be given in a dose of 0.5 mg every 24 hours or 0.1 Gm every 6 hours.

#### Toxicity

Occasionally omitting diarrhoea, stomatitis and dermatitis occur orally. Hypotension may result from prolonged intravenous treatment at high doses. Thrombophlebitis may result from intravenous administration. Superinfection with resistant staphylococci may occasionally occur.

## TETRACYCLINE (ACHROMYCIN® TETRACYN®)

Tetracycline is produced by removing the chloro from chlorotetracycline. Its properties are closely similar to those of chlorotetracycline and oxytetracycline. It is more stable in solution than either derivative.

Indications and Anti Infective Spectrum (See table on p 314)  
 Tetracycline is a broad spectrum antibiotic whose field of activity is similar to those of chlorotetracycline and oxytetracycline. Susceptibility of strains of bacteria may differ among the three drugs, however.

#### Absorption and Excretion

Tetracycline is well absorbed and excreted similarly to chlorotetracycline. It may differ more readily into the cerebrospinal fluid.

#### Dose and Route of Administration

- A Oral 0.25 to 1.0 Gm every 6 hours  
 B Intravenous 0.5 to 1.0 Gm every 12 hours  
 C Intramuscular 0.1 Gm every 8-12 hours

## 510 Chloramphenicol Tyrothricin Polymyxin

### Toxicity

Similar to that of chlortetracycline and oxytetracycline but significantly less frequent

## CHLORAMPHENICOL (CHLOROMYCETIN®)

Chloramphenicol (Chloromycetin®) originally prepared from *Streptomyces venezuelae* is also produced synthetically

Indications and Anti Infective Spectrum (See table on p. 514)  
Chloramphenicol is active against a wide range of bacteria, the rickettsiae and the viruses of lymphopatia venereum, psittacosis and primary atypical pneumonia. Generally speaking, it is more effective than chlortetracycline and oxytetracycline in typhoid fever approximately equal in effect against other gram negative organisms, spirochetes and rickettsiae.

### Absorption and Excretion

Chloramphenicol is rapidly absorbed from the gastrointestinal tract, reaching a peak serum concentration within 3 hours. Absorption following rectal administration is slightly less efficient. 0.5 Gm. may be administered intramuscularly or intravenously every 6 hours. Excretion is principally by the kidneys, and high concentrations are reached in the urine.

### Dosage and Route of Administration

- A Oral, 50 mg /kg (23 mg /lb) per day (100 mg /kg or 45 mg /lb per day in children) administered every 3 to 6 hours. Reduce by one half following defervescence.  
B Rectal, 125 to 150 mg /kg (56 to 70 mg /lb) per day in children every 6 hours. Capsule should be punctured before insertion.  
C Intramuscular or Intravenous, 500 mg every 6 hours.

### Toxicity

Nausea and vomiting, diarrhea, nervous depression and dermatitis occur occasionally. Granulocytopenia and aplastic anemia occur occasionally, and chloramphenicol should therefore be used only on definite indication.

## TYROTHRICIN

Tyrothricin is prepared from *Bacillus brevis*. It is used topically as an ointment or watery suspension. It is active only against gram positive organisms. Because of toxic effects on peroral administration, its use is limited entirely to the topical treatment of infected wounds and pyoderma.

## POLYMYXIN (AEROSPORIN®)

The polymyxins, of which B, D, and E have been given clinical trial, are derived from *Bacillus polymyxa* and related organisms.

Indications and Antimicrobial Spectrum (See table on p. 514)

With the exception of most strains of *Proteus vulgaris*, polymyxin is bactericidal against gram-negative rods. Most strains of *Pseudomonas aeruginosa* (pyocyanus) are highly susceptible to polymyxin. Polymyxin is indicated in severe systemic infections due to gram-negative rods, particularly infections due to *Pseudomonas aeruginosa* which do not respond to other forms of chemotherapy. It may be used as a local application in wounds infected with susceptible organisms. It may be given orally in the treatment of shigellosis or in the management of the carrier state.

Administration

Absorption is rapid after intramuscular injection. Excretion is largely by the kidney, and high concentrations are achieved in the urine. Polymyxin is not absorbed from the gastrointestinal tract, and when it is given by mouth it exhibits its principal activity in the lumen of the bowel.

Dosage

- A. Intramuscular: 1.5 to 2.5 mg /Kg of body wt. divided into 3 or 4 doses.  
 B. Oral: 20 mg /Kg of body wt. /day given in 3 or 4 doses.

Toxicity

Mortality is of the order of dosage levels over 2.5 mg /Kg of body wt. /day. Albuminuria and nitrogen retention are usually reversible. Weakness, drowsiness, ataxia, numbness of the fingers and feet, impaired position sense, blurring of the vision, diplopia, and nystagmus may occur. Allergic reactions such as itching, chilly sensations, sneezing, and rash have been observed. Irritation at the site of intramuscular injection is common.

**BACITRACIN**

Bacitracin is derived from the growth products of *Bacillus*.

Indications and Antimicrobial Spectrum (See table on p. 514)

Bacitracin is active against many positive cocci and spirochetes. Its action is principally bactericidal. Synergistic action with penicillin, and other bactericidal antibiotics has been demonstrated against staphylococci and other organisms. Bacitracin is principally used orally for local infections due to susceptible organisms, but it may be used parenterally in the treatment of infections resistant to other antibiotics or in combination with other antibiotics as one of a synergistic pair. It may be used locally in the treatment of smudge colitis.

Dosage

- A. Injection: Solution or suspensions containing 500 units per cc.  
 B. Local: 40,000 to 120,000 units in divided doses daily for 3 to 20 days.  
 C. Intramuscular: 2500 to 25,000 units every 8 hours.

Toxicity

Albuminuria cylindruria and nitrogen retention commonly occur after parenteral administration

**NEOMYCIN**

Neomycin is derived from *Actinomyces fradit*

Indications and Antimicrobial Spectrum (See table on p 514 )

Neomycin is most active against gram negative rods but is also active against many strains of gram positive cocci particularly staphylococci as well as gram positive rods. Many strains of *Proteus vulgaris* are sensitive to neomycin. Its principal use is local in the treatment of infections due to susceptible organisms but it may be used occasionally parenterally in the treatment of infections due to organisms resistant to other antibiotics or may be used as one of a synergistic pair. It may be used orally to sterilize the bowel before gastrointestinal surgery and in amebic colitis.

Absorption and Excretion

Neomycin is readily absorbed after intramuscular injection. It is poorly absorbed from the gastrointestinal tract. It exerts its principal activity in the lumen of the bowel when given orally. Neomycin is principally excreted by the kidney and appears in the urine in high concentration.

Dosage

- A Topical Ointments containing 1000 units per gram or solutions containing 200 units per cc. may be used locally.
- B Oral 50 000 units every 6 hours.
- C Intramuscular 100 000 units to 200 000 units daily divided into doses at 6 hour intervals.

Toxicity

Renal damage manifested by albuminuria. Nitrogen retention may occur. Deafness may follow parenteral administration.

**ERYTHROMYCIN (ERYTHROCYN®)**

Erythromycin is a medium spectrum antibiotic derived from *Streptomyces erythraeus*. Its action may be bactericidal or bacteriostatic depending on the susceptibility of the bacteria. Resistance to erythromycin may develop rapidly under certain circumstances most notably by staphylococci.

Indications and Antimicrobial Spectrum (See table on p 514 )

Erythromycin is active against most strains of gram positive cocci gram negative cocci *C. diphtheriae* *H. influenzae* *B. pertussis* and brucellae. Activity has also been shown against the viruses of lymphopathia venereum and psittacosis and the rickettsia of typhus. Erythromycin may be used in infections due to these organisms as an alternative to penicillin and other antibiotics.

Route of Administration and Dosage

A	Oral	0.2 to 0.5 Gm	every 6 hours
B	Intravenous	0.5 Gm	every 12 hours

Toxicity

Nausea, vomiting and diarrhea occur occasionally

**CARBOMYCIN (MAGNACYCIN®)**

Carbomycin is a medium spectrum antibiotic derived from *Streptomyces halstedii*. It is principally active against gram positive cocci but is also active against large virus and rickettsiae and *E. histolytica*.

The indications for carbomycin are not established. It may exert therapeutic effect in infection of the hepatic biliary tract by susceptible organisms including *E. histolytica*.

Absorption and Excretion

Carbomycin is absorbed from the gastrointestinal tract and excreted promptly in high concentration in the bile. Little appears in the serum or urine.

Dosage and Indications

1	Oral	0.5 Gm	every 6 hours
2	Intravenous	0.5 Gm	every 12 hours

**TABLE OF SUSCEPTIBILITY OF MICROBIAL PATHOGENS  
TO CHEMOTHERAPEUTIC AGENTS**

Data are principally based on available clinical experience and to a lesser extent on *in vitro* data. The drug of best clinical use may be supplemented if other experience

Drug of choice	■ No significant activity	(3) Sulfoxes (Prometle® Promile®)
Alternative drug	▼ Significant variation in susceptibility of strains	C Combined therapy
No activity or inactivation due	↑ If data available	

[illegible]

## Chapter 20

# DISEASES OF UNKNOWN ETIOLOGY

A variety of names (collagen diseases, diffuse vascular diseases, visceral angitides, diffuse connective tissue disease) have been given to a group of diseases which appear to have in common a pathological involvement of mesenchymal tissues. Rheumatic fever, rheumatoid arthritis, disseminated lupus erythematosus, periarthritis nodosa, scleroderma, dermatomyositis, acrocyanosis (scleroderma) and perhaps glomerulonephritis are the chief members of this group of rather ill defined but probably related diseases of unknown etiology. The differentiation of these disorders sometimes poses a real clinical problem, and in many instances the diagnosis can be established only after long continued and painstaking observation (see page 320). There is some evidence that hypersensitivity is common denominator in etiology, although the pathological reaction in the connective tissue is probably caused by a wide variety of injurious agents.

### Clinical Findings

Certain clinical features are common to many of the collagen diseases although there may be considerable variation in the severity, extent and frequency of manifestations.

- A. Cutaneous Lesions. But chiefly to be seen: erythema multiforme and nodosum.
- B. Hemorrhagic Phenomena. Purpura, hemorrhage.
- C. Articular Manifestations. Symmetrical arthritis, arthralgia.
- D. Cardiac Involvement. Pericarditis, myocarditis, myocardial coronary artery disease.
- E. Vascular Involvement. Arterial hypertension, Raynaud's phenomenon.
- F. Lymphatic Involvement. Lymphadenopathy and splenomegaly.
- G. Immunologic Reactions. Polyserositis, pleurisy, pericarditis, peritonitis.
- H. Neurological Manifestations.
- I. Hematologic Changes. Anemia, leukocytosis or leukopenia.
- J. Renal or Urinary.
- K. General Constitutional Manifestations. Fever, weight loss, fatigue.

### Laboratory Findings

Laboratory studies which often are of special diagnostic significance:

- A. Serology. Characteristic histologic changes may be observed in some of these conditions.



- B Muscle Biopsy.** Characteristic histologic changes occur in periostitis and dermatomyositis
- C Lupus Erythematosus C II Demonstration.** These characteristic polymorphonuclear leukocytes (with round vacuole partially filled with nuclear material) in marrow and peripheral blood smears point relatively specifically to disseminated lupus
- Antistreptolysin Titer.** Demonstration of changing A S titer may provide etiologic information regarding background of previous streptococcal infection which is of especial value in the diagnosis of rheumatic fever

Rheumatic fever and Sydenham's chorea will be discussed individually. The treatment of other diffuse collagen diseases will be discussed collectively. Acrocyanosis (acrosclerosis) has been included in this group for purposes of differential diagnosis although it is doubtful that it is a true member of the collagen disease group. Rheumatoid arthritis has been dealt with in Chapter III (see page 311) glomerulonephritis in Chapter II (see page 283)

### RHEUMATIC FEVER (code No 010 932)

A generalized disease of unknown etiology usually coming on 1-3 weeks after an acute infection with the hemolytic streptococci and manifested usually by pathological changes involving the heart blood vessels and serous surfaces primarily the joints. It has a marked tendency to recur.

#### Diagnosis

The diagnosis of active rheumatic fever can usually be made if the patient has 2 major manifestations or 1 major and 2 minor manifestations of the disease (Jones)

##### A Major Manifestations

- |  |                          |
|--|--------------------------|
| 1 Definite past history of rheumatic fever         | 3 Inflammation of joints |
| 2 Signs of active carditis (including Ecg changes) | 4 Subcutaneous nodules   |
|  | 5 Chores                 |

##### B Minor Manifestations

- |                       |                            |
|-----------------------|----------------------------|
| 1 Fever               | 5 Non traumatic nose bleed |
| 2 Erythema multiforme | 6 Purpura                  |
| 3 Abdominal pain      | 7 Pneumonitis              |
| 4 Precordial pain     |                            |

- Laboratory Tests.** May show increased sedimentation rate and increased antistreptolysin titer leukocytosis and anemia and abnormal Ecg

#### Treatment (See page 168 for treatment of rheumatic heart disease)

##### A Specific Measures

- 1 Salicylate therapy.** The salicylates markedly reduce fever alleviate joint pain and possibly reduce joint swelling. There is no evidence that they have any effect on the natural course of the disease. The salicylates should be continued as long as necessary to relieve pain swelling or fever. If withdrawal of the drug results in a recurrence of symptoms they should immediately be reinstituted.

- a. Sodium salicylate is the drug most widely used -

(1) Dose 1-2 Gm (15-30 gr) every 3-4 hours orally  
The drug should be given in sufficient doses to allay symptoms and fever and if necessary to give maximum doses to achieve this result. In an occasional patient maximum doses may not be completely effective. There is no evidence that intravenous administration has any advantage over the oral route.

(2) Toxic reactions. The early reactions include tinnitus, nausea and vomiting. Sodium salicylate may be given in enteric-coated 0.5 Gm (7½ gr) pills or with small doses of sodium bicarbonate to reduce gastric irritation. Never use sodium salicylate or sodium bicarbonate in patients with acute rheumatic fever who have associated cardiac failure.

- b. Acetylsalicylic acid may be substituted for sodium salicylate with the same dosage and precautions.

c. Aminopyrine (pyramidon). If the salicylates are not tolerated this drug may be used in doses of 0.3-0.4 Gm (3-4 gr) every 3-4 hours. Check the WBC every 3-4 days when giving this drug.

2. The sulfonamide and penicillin should never be used in the treatment of acute rheumatic fever; they are of no value and the sulfonamides may be harmful.

a. Prevention of relapse - There is some evidence that penicillin may prevent a relapse if used within 2-4 hours after the onset of a streptococcal infection.

b. Coinciding infection. If an acute infection occurs during an attack of rheumatic fever give antibiotic as indicated (see page 314) but avoid giving sulfonamides.

## B. General Management

1. Bed rest should be enforced until all signs of acute rheumatic fever have disappeared. The criteria for this are:

- Return of the temperature to normal with patient in bed rest and without medications.

b. Normal sedimentation rate.

c. Normal resting pulse rate (under 100 in adults).

d. Return of ECG to normal or fixation of abnormalities.

2. Gradual resumption of activities. Patient may be allowed up slowly but several months should elapse before return to full activity unless the infection was exceedingly mild.

3. Maintain good nutrition.

C. Cortisone, Atrial and ACTH. Although rather remarkable results have been observed in certain acute rheumatic fever patients treated with these drugs, such improvement is only temporary. There may be a prompt disappearance of fever, malaise, tachycardia, and polyarthritides. Abnormal ECG changes (prolonged P-R interval) and blood enzyme titer may return to normal limits within a week. Optimal dosage schedules and influence of the drugs on the development of subsequent cardiac lesions have not been established.

## D. Treatment of Complications

1. Compensated Congestive Failure. See also for congestive failure (see page 112), with the following variations:

- a Low sodium diet (see page 53) and mercurial diuretics (see page 204) are of particular value in promoting diuresis and treating failure in acute rheumatic fever
  - b Digitalis is generally not as effective in acute rheumatic fever as in most cases of congestive failure and the drug may accentuate the myocardial irritability producing arrhythmias that further embarrass the heart
  - c Many cases of congestive failure are due to acute myocarditis. These cases often respond dramatically to corticotropin (ACTH) or cortisone. When these agents are used for this condition maximal sodium restriction (under 200 mg daily) is imperative
- 2 Pericarditis. Treat as any acute non purulent pericarditis (see page 188). The rheumatic effusion is sterile and antibiotics are of no value. The general principles include relief of pain by opiates if necessary and removal of fluid by cardiac paracentesis if tamponade develops. If paracentesis is performed it should be preceded and followed by a short course of penicillin therapy to prevent contamination of the pericardium. ACTH and cortisone as well as salicylates should be continued or started as they seem to have a specific and favorable effect in aiding resorption of the fluid.

### Prophylaxis

The principles of prophylaxis are to avoid hemolytic streptococcal infection and to give immediate treatment with the antibiotic agents if a streptococcal infection occurs.

#### A. General Measures

- 1 Avoid contact with persons who have colds or upper respiratory infections
- 2 If possible live in a warm climate

#### B. Prevention of Infection. Two methods of prophylaxis are now advocated

- 1 Penicillin. Oral penicillin in doses of 100 000 units t i d (under 100 lbs weight) 200 000 units t i d (over 100 lbs weight). This is advocated especially for children who have had one or more attacks of acute rheumatic fever and should be given throughout the school year. Adults should receive this for about 5 years after an attack of acute rheumatic fever. In any case it should be given to these individuals between September and June.
- 2 Sulfonamides. If penicillin is not available give sulfadiazine 1-2 Gm (15-30 gr) daily in divided doses from September to June. Patients receiving sulfonamides should have frequent blood counts; urinalysis should be performed initially and at least every 4-6 weeks thereafter. If there is any tendency towards leukopenia the drug should be stopped immediately.

C. Treatment of streptococcal sore throat should be carried out by use of the antibiotics. It has been shown that prompt therapy (within 24 hours) of streptococcal infections by 300 000 600 000 units of aqueous procaine penicillin I M will prevent most attacks of acute rheumatic fever. If this proves true prompt penicillin treatment of any upper respiratory infection in susceptible individuals may be adequate prophylaxis.

## CHOREA (Sydenham W) (code No 930 195)

As a common manifestation of rheumatic disease characterised pathologically by generalized demyelination and congestion of the brain and by involvement of the basal ganglia with arterial thrombi focal hemorrhages perivascular infiltration and chromatolysis of nerve cells. It occurs most frequently in females in the second decade and is characterized by jerky restless choreiform movements and often by dysarthria.

Treatment

- A Specific Measures None known. Corticotropin (ACTH) and cortisone appear to be of benefit in many cases of chorea. It must be given in relatively high initial dosage and marked sodium restriction must be employed.
- B For the Symptom may be employed if all else fails. This may be given by the use of 2 methods: Hyperthermia apparatus with temperature 39.5-40.5°C (103-105°F) for 3-5 hours twice weekly for 6-10 treatments or typhoid vaccine in 1 V daily for 3-7 days.

C General Measures

1. General symptoms: attention and good nursing care are most important.
2. Sedation with Phenobarbital: Phenobarbital 15-30 mg (1/4-1/2 gr) t.i.d. q.i.d. may be helpful.
3. If convulsive seizures appear magnesium sulfate 4-10 cc (1-2 1/2 cc) of a 10% solution I.M. or I.V. may be used. When administering magnesium sulfate I.V. always have a syringe filled with 10 cc of 10% solution of calcium gluconate or other calcium salt ready to administer I.V. if nausea or urticaria or other allergic reaction occurs.

OTHER DISEASES OF UNKNOWN ETIOLOGY  
OTHER VISCERAL AGITATIONS (Diffuse Vascular Diseases)

Acrocyanosis (code No. not listed)

Dyserythema (code No. 114 311)

Disseminated lupus erythematosus (code No. 118 150)

Dermatomyositis (code No. 2 4 100)

Periostitis nodosa (code No. 440 190)

Diagnosis

See label on page 320

Treatment

- A Specific Measures None known. The role of corticotropin (ACTH) and cortisone in the management of this group of diseases has not yet been established, although they seem to be the most effective weapons available. In some cases the effect has been quite definite and dramatic, especially in the erythema of the limbs. Other patients have received temporary benefit during acute episodes. A few patients seem to be only intermittently affected by these groups. Suggested doses are comparable to those employed in rheumatoid arthritis (see page 311).

## DISEASES DUE TO PHYSICAL AGENTS

### DISORDERS DUE TO COLD

Exposure to cold produces immediate localized and then generalized vasoconstriction. When the skin temperature falls to 25° C (77° F) tissue metabolism is slowed but the demand for oxygen remains greater than the slowed circulation can supply and the area becomes cyanotic. At 15° C (59° F) tissue metabolism is markedly decreased and the dissociation of oxyhemoglobin is reduced giving a pink well oxygenated appearance to the skin. Evidence indicates that tissue survival at this temperature is slight. Tissue death may be caused either by ischemia and thromboses in the smaller vessels or by actual freezing with the formation of ice in the tissues. Freezing does not occur until the skin temperature drops to -4 to -10° C (23 to 14° F) or even lower depending on coexisting factors such as wind, immobility, venous stasis, malnutrition and occlusive arterial disease.

#### Prophylaxis

1. Wear warm, dry clothing preferably several layers to afford additional insulation with windproof outer garment.
2. Keep dry when possible; remove wet clothing, socks and shoes and replace with thoroughly dried ones.
3. Avoid cramped positions, constricting clothing, and prolonged dependency of feet.
4. Exercise arms, legs including fingers and toes periodically to maintain circulation.
5. Avoid wet and muddy ground and keep sheltered from wind.
6. Maintain good nutrition and cleanliness of skin.

### CHILBLAINS (code No. 0 -446)

Chilblains are red, itching skin lesions usually on the extremities caused by exposure to cold without actual freezing of the tissues. They may be associated with edema or blistering and are aggravated by the application of warmth. In the chronic form ulcerative or hemorrhagic lesions may appear and progress to scarring, fibrosis and atrophy.

#### Treatment

1. Protect affected area from trauma and secondary infection.
2. Do not rub or massage injured tissues or apply ice or heat.
3. Elevate affected part slightly and allow to warm gradually.

Frostbite is a injury of the superficial tissues due to freezing. It may be divided into three grades of severity. 1st degree Frostbite without blistering or peeling. 2nd degree Frostbite with blistering or peeling. 3rd degree Frostbite with death of skin and underlying tissues. In mild cases the numbness persists and it may be painful. In severe cases the tissue may become necrotic and it may be necessary to amputate. Frostbite may be prevented by wearing warm clothing and avoiding exposure to cold. Frostbite may be treated by rewarming the affected area and by using painkillers. Frostbite may be prevented by wearing warm clothing and avoiding exposure to cold. Frostbite may be treated by rewarming the affected area and by using painkillers.

Treatment  
The objective of treatment is to prevent further tissue damage and to promote healing. Frostbite should be treated by rewarming the affected area. This may be done by immersing the limb in warm water (40-42°C) or by using a heating pad. Frostbite should be treated by rewarming the affected area. This may be done by immersing the limb in warm water (40-42°C) or by using a heating pad.

Prevention  
Frostbite may be prevented by wearing warm clothing and avoiding exposure to cold. Frostbite may be prevented by wearing warm clothing and avoiding exposure to cold. Frostbite may be prevented by wearing warm clothing and avoiding exposure to cold. Frostbite may be prevented by wearing warm clothing and avoiding exposure to cold. Frostbite may be prevented by wearing warm clothing and avoiding exposure to cold.

First Aid  
1. Protection of the local part.  
2. Avoid exposure to the cold.  
3. Keep affected part warm.  
4. Avoid direct contact with the frost.  
5. Avoid rubbing the affected part.  
6. Avoid use of alcohol.  
7. Avoid use of tobacco.  
8. Avoid use of drugs.  
9. Avoid use of cosmetics.  
10. Avoid use of jewelry.

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4 Treat shock when present (see page 31)

II Follow up Avoid immediate re exposure to heat

### HEAT CRAMPS (code No 270-445)

Heat cramps are painful spasms of voluntary muscles of abdomen and extremities due primarily to salt depletion and following sustained exposure to heat. The skin is moist and cool and muscle twitchings may be present. The temperature is normal or only slightly increased. Laboratory studies reveal hemoconcentration and low serum sodium.

#### Treatment

##### A Emergency Measures

1 Sodium chloride 1 Gm (15 gr) every 1/2 hour with abundance of water or saline solution by mouth or 1000 cc (1 qt) physiological saline I V. This usually relieves attack promptly.

2 Have patient rest in a cool, shady place.

3 Massage sore muscles gently.

II Follow up Rest for 1-3 days depending on severity.

### BURNS

Burns are tissue injuries due to heat and may be graded as follows:

1st degree: Erythema without blistering (code No 13-4411)

2nd degree: Erythema with blistering (code No 13-4412)

3rd degree: Destruction of deeper tissues (code No 13-4413)

When tissues are burned, plasma is lost into the burned area and from the surface of the burn. This leads to hypoproteinemia which remains as long as a granulating surface is present and the granulating surface heals poorly as long as there is hypoproteinemia. The loss of plasma results in a reduced blood volume, hemoconcentration, low cardiac output, decreased blood flow, oliguria, elevated N.P.N. and leukocytosis. Though anemia may occur at the time of the burn due to rbc destruction, it more commonly becomes apparent about the fifth day as hemodilution begins and as the effects of blood destruction and impaired rbc formation make themselves apparent. Secondary infection is prone to occur and must be treated promptly. Death may result in an adult when 30% or more of the body surface is involved. In an infant, 10% may be associated with very severe effects.

The course of a severe burn may be divided as follows:

- 1 Immediate shock (neurogenic)
- 2 Burn shock (first 48 hours)
- 3 Toxemia (occurring about 3rd day)
- 4 Sepsis (about 3rd day)
- 5 Healing and restoration of function

#### Treatment

Take blood pressure, pulse, hemoglobin, RBC hematocrit and plasma proteins at start of therapy and at regular intervals.





for several weeks sloughing then occurs slowly and in a fairly wide area. Electric shock may produce loss of consciousness which may be momentary or prolonged. With recovery there may be muscular pain, fatigue, headache, and nervous irritability. The physical signs vary according to the action of the current. With ventricular fibrillation no heart sounds or pulse can be found and patient is unconscious. The respirations continue for a few minutes becoming exaggerated as asphyxia occurs and then ceasing as death intervenes. With respiratory failure respirations are absent and the patient is unconscious, the pulse can be felt but there is a marked fall in blood pressure and the skin is cold and cyanotic.

### Treatment

#### A Emergency Measures

1. Free from current at once. This may be done in many ways but rescuer must protect himself in the process. Throw the proper switch, sever the wire with a wooden handled axe, make proper ground to divert current, or drag victim away by means of dry clothing or leather belt as indicated.
2. Artificial respiration must be started immediately (see page 151) if victim has slow or absent breathing and continued until spontaneous breathing returns or rigor mortis sets in.
3. Precordial compression for ventricular fibrillation or arrest. Artificial respiration will not restore its normal beat and other measures may not either.
4. Treat shock promptly (see page 31).
5. Positive pressure oxygen with carbon dioxide may be used when available or oxygen and carbon dioxide by mask combined with artificial respiration.

#### B Hospital Measures

1. Hospitalize patient when required and observe for sudden cardiac dilatation or secondary hemorrhage.
2. Lumbar puncture if signs of increased pressure are noted.
3. Treat burns conservatively. The direction and extent of tissue injury may not be apparent for weeks. Infection is usually not a problem early. Patience and delay are important in treatment, allow granulation tissue to be well established before attempting any surgery. Hemorrhage may occur late and may be severe.

## IRRADIATION SICKNESS

Irradiation sickness is the term applied to the syndrome developing during or after the course of therapeutic x-ray administration or after exposure to ionizing radiation (e.g., x-rays, ultraviolet gamma rays, alpha or beta particles).

### Irradiation Sickness Associated with Irradiation Therapy

Anorexia, nausea, vomiting, weakness, exhaustion, lassitude, and in some cases prostration may occur singly or in combination and may be of varying severity. The symptom complex is most likely to occur when x-ray therapy is given over the upper abdomen, less often over lower abdomen or thorax, and rarely over the extremities.



## Chapter 22

# DISEASES DUE TO TOXINS

### PRINCIPLES OF TREATMENT OF ACUTE POISONING

In the emergency treatment of any poisoning in which the toxin has been taken by mouth the following general procedures should be carried out

- A Remove poison by emesis lavage catharsis or diuresis as soon as possible
- B Inactivate poison with specific or general antidote Follow with lavage
- C Combat shock collapse and specific manifestations as they arise
- D Protect mucous membranes with demulcents

#### Removal of Poison.

Do not use stomach tubes or emetics in poisonings due to strong acids or alkalis or other corrosive agents they may cause gastric perforation

- A Emesis. This is the quickest way to evacuate gastric contents

##### 1 Indications

- a For removal of excess poison in cooperative patients
- b Convenience When stomach tube is unavailable or patient is unable to take stomach tube

##### 2 Contraindications

- a For drowsy or unconscious patients Danger of aspiration of stomach contents
- b For patients who have swallowed corrosive poisons

##### 3 Technic

- a Introduction of finger feather or other object into throat
- b Drug or chemical Give one of the following and follow with copious quantities of warm water ■ neals should be continued until gastric contents are clear

(1) Apomorphine hydrochloride 8 mg ( $\frac{1}{10}$  gr) subcut will often quiet the patient and will usually induce vomiting

(2) Mustard powdered 1-3 tsp in a glass of lukewarm water ■ an uncertain and unpleasant emetic but is often useful and has the advantage of being generally available

(3) Sodium chloride 1 Tbsp in a glass of lukewarm water ■ not very efficient but is readily available

(4) Strong soapuds 250-500 cc ( $\frac{1}{2}$  - 1 pt)



**COMMONLY EMPLOYED AGRICULTURAL POISONS  
AND TREATMENT OF POISONING BY THESE AGENTS**

Chemical Type	Examples	Treatment
Metals	Arsenicals Calcium arsenate lead arsenate copper acetarsenite (Paris green)	HAL® therapy (See page 536)
	Lead salts Lead arsenate	Calcium EDTA (See page 542) DO NOT USE HAL®
	Copper salts Copper sulfate (blue vitriol) as used in Bordeaux mixture	Symptomatic and supportive Sodium thio-sulfate may be of value (See page 540)
Fluorides	Sodium fluosilicate sodium fluoroacetate (1080) sodium fluoride	Symptomatic and supportive Barbiturates may be of value in controlling symptoms of CNS excitation (See page 533.) Symptomatic and supportive
Halogenated hydrocarbons	Dichloro diphenyl trichloro ethane (DDT) chlorinated camphene (Toxophene) dichlorophenoxy acetic acid (2,4,5) benzene hexachloride ethylene dibromide methyl bromide chlordan methoxychlor	
Thiocyanates	Lethal cyanide	Parenteral administration of large doses of atropine sulfate and magnesium sulfate appears to relieve the marked neuro muscular toxicity Give oxygen by positive pressure if pulmonary involvement develops (See page 148)
Organic phosphate esters	Heptachlor triphosphate (Hi-TP) tetraethylpyrophosphate (TEPP) parathion diethylthio phosphate (Parathion) disopropyl fluorophosphate (DFP)	(See page 540) <i>rapidly fatal</i> Treat promptly Symptomatic and supportive
Cyanides	Hydrocyanic acid gas (cyanogen and cyanogen gas) and cyanide salts	Maintain adequate ventilation using artificial respiration and oxygen if necessary
Thiourea compounds	Alphanaphthylthiourea (ANTU)	Symptomatic and supportive
Plant derivatives	Nicotine (tobacco or Nicotiana glauca)	Symptomatic and supportive
	Rot none and pyrethrum	

## A C1 1 story Fluor

- 1 Shock (see page 32) Principal are incised recumbent position warmth administration of stimulants and paren-  
tally fluid to incise as of the blood volume  
2 Cardiac failure (see page 182) Principal measures include  
oxyg and digitaline and rarely intra-  
cardiac adrenaline  
3 Pulmonary edema Principal measures include oxyg and  
parentally by position (see page 142) o treatment  
to digitaline if it exists avoid administration of pa-  
rental saline or other parenteral fluid (except plasma)

## B R 1 story Abnorm Illi

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- 1 **CNS depression** Use stimulant (analeptic) drugs (see previous page)
- 2 **Dehydration** Use oral or parenteral fluids as tolerated and indicated (see page 23)
- 3 **Pain** See analgesic and narcotic drugs on pages 36 and 37

### ACIDS CORROSIVE (code No 010-32)

The strong mineral acids exert primarily local corrosive effect on the skin or mucous membranes. In severe burns circulatory collapse may result. The M L D is 4 cc (1 dr) of concentrated acid. Symptoms include severe pain in throat and upper gastro intestinal tract, marked thirst, bloody vomitus and difficulty in swallowing, breathing and speaking. There is discoloration and destruction of skin and mucous membranes in and about mouth collapse and shock.

#### Treatment

1. Lime water, magnesia or aluminum gel orally to neutralize acid. Avoid carbonates or bicarbonates internally if possible since these form gas and cause distension of already weakened stomach wall.
2. Avoid emetics or lavage if perforation is a possibility.
3. Egg whites beaten with 500 cc (1 pt) milk or water as demulcent.
4. External burns. Wash with water and sodium bicarbonate.
5. Eyes. Wash well with 1% sodium bicarbonate solution.
6. General supportive care as indicated (see page 331).

### ALCOHOL, ETHYL

Ethyl alcohol is a mucous membrane irritant and a CNS depressant. The M L D is 100-200 cc (3-7 oz) of pure alcohol when ingested at one time.

#### Treatment

##### A Acute Intoxication (code No 010-332)

1. Careful physical examination especially for evidences of head injury.
2. Avoid sedatives or narcotics.
3. Lavage stomach with lukewarm water containing 4 Gm (1 tsp) sodium bicarbonate or give apomorphine 4 mg (1/10 gr) if alcohol has recently been ingested.
4. Stimulants
  - a. Strong black coffee orally or rectally.
  - b. Caffeine sodium benzoate 0.2-0.5 Gm (3-7 1/2 gr) every 4 hours subcut.
  - c. Coramine® 1-5 cc of 25% solution I.V.
5. Oxygen therapy for comatose patients if needed. Immediate cause of death is usually respiratory failure.

##### B Delirium Tremens (code No 003-332) (see page 337)

## ALCOHOL, METHYL (code No 010 331)

Methyl alcohol is a caustic and irritant and CNS depressant which has an affinity for the optic nerve. It is slowly excreted from the body and is metabolized giving formaldehyde and formic acids and products which produce acidosis. The M.L.D. by ingestion is 30-80 cc (1-2 oz). The pathologic experience is a headache, tripping in dyspnea, nausea and vomiting and may result in loss of vision. Examination reveals hyperemia, cyanosis, ecchymosis or depression, delirium, coma and convulsions.

Treatment:

1. Large amounts well with 1 or 2% sodium bicarbonate
2. Check serum CO<sub>2</sub>
3. Sodium bicarbonate 0.5 Gm (1 1/2 gr) orally every 2 to 3 hours as necessary or 100-300 cc 5% NaHCO<sub>3</sub> i.v. (5 page 24)
4. Keep patient in dark room and institute supportive measures as indicated.

## ALKALIS (code No 010-32)

Tris in household are common ingredients of household cleaning compounds and may accidentally be ingested. They exert their effects on the skin and mucous membranes but in instances of severe involvement it may cause circulatory failure and collapse. The M.L.D. for the caustic powders (NaOH and KOH) is 15 Gm (1/2 oz). For strong ammonia water 4 cc (1 dr). Recovery has followed much larger doses, however. There is symptom of burning pain in upper gastrointestinal tract as well as vomiting, and difficulty in swallowing and breathing. Physical examination reveals dehydration and edema of skin and mucous membranes in and about mouth, bloody sputum and stool, dyspnea, and is watery collapse.

Treatment:

1. A mild emetic and lavage if perforation is a possibility
2. Dilute ingestion of strong alkali is to neutralize alkali 120 to 240 cc (1/2 to 1 oz) of 0.5% hydrochloric acid may be used
3. Give fat of a salad oil (helps neutralization by formation of soaps), whites of 4 eggs well mixed with water or 1-2 Theophrastus gelatin 100 cc (1 pint) of water
4. Watch for edema of the lungs and a heartening if needed
5. Supportive measures as necessary
6. Wash of nail burns with dilute sugar or citric acid
7. Wash eyes with boric acid solution or water

## ARSENIC

(Acute: code No. 010 3114) (Chronic: code No. 011 3114)

Arsenic is found in industrial chemicals, eg. brucine, for years and dusts, rodenticides, household ant poisons and arsenical medications.



General Reaction

Any of the following may be present: severe nausea and vomiting; abdominal cramps; diarrhea; marked thirst; choking sensation; and difficulty in swallowing; cyanosis; marked cerebral symptoms; and coma.

Treatment

1. Emetic or abundant gastric lavage with warm water.
2. Follow with demulcent drink.
3. Symptomatic relief of diarrhea (e.g., codeine).
4. Dimercaprol Injection U.S.P. (BAL®) 10% solution in oil. Give 1 M (Eagle J. Ven. Dis. Inform. 37:114, 1946).
  - a. Severe poisoning: 3 mg/Kg/dose (1.8 cc/80 Kg)
    - 1st day: 1 injection every 4 hours d y and night
    - 2nd day: 1 injection every 4 hours day and night
    - 3rd day: 1 injection every 6 hours for 4 doses
    - 4th day on: 1 injection b i d for 10 days or until recovery is complete
  - b. Mild poisoning: 2.5 mg/Kg/dose (1.5 cc/80 Kg)
    - 1st day: 1 injection every 4 hours for 4 doses
    - 2nd day: 1 injection every 4 hours for 4 doses
    - 3rd day: 1 injection b i d
    - 4th day on: 1 injection once or twice a day for 10 days or until recovery is complete
  - c. Toxic reactions to BAL®: These appear to be transient and over in 30 minutes. They include nausea, vomiting, headache, generalized aches and pains, and burning sensation about the head and face. Barbiturates have been recommended for severe side effects.

**BARBITURATES (code No. 010 3371)**

Barbiturates are used for sedative, hypnotic or anticonvulsant purposes. The barbiturates are one of the most common means of both suicidal and accidental poisoning.

Obtain data on dosage and time of ingestion from patient, relatives, friends or attending physician when possible.

- A. Mild Symptoms: Drowsiness, mental confusion, headache; there may be euphoria or irritability.
- B. Moderate or Marked Symptom: Delirium, stupor, shallow and slow respirations, circulatory collapse, cold clammy skin, cyanosis, pulmonary edema, dilated and non-reacting pupils, hyporeflexia, areflexia, coma, and finally death.

Treatment

- A. Mild Symptoms: Symptomatic and supportive nursing care. Stimulants should be limited to caffeine. Keep patient under observation until danger has passed. Place suicidal patients under psychiatric care.
- B. Moderate or Marked Symptoms
  1. Hospitalize.
  2. Combat shock (see page 32).
  3. Record the following observations at 15 to 30 minute intervals until danger has passed:

- a Temperature pulse respiration and blood pressure
  - b Mental status or state of consciousness
  - c Skin color (cyanosis or pallor)
  - d Lung bases (pulmonary edema)
  - e Reflexes (corneal, pupillary, gag, plantar)
  - f Sensation (response to pin)
- 4 Gastric lavage with 1:2000 solution potassium permanganate  
Be certain to move all potassium in final aspiration  
This is of doubtful value if performed more than 6 hours  
after ingestion of the drug and may be very dangerous  
CAUTION Danger of aspiration pneumonia is great in  
stuporous or comatose patient
- 5 Pupilation of no value
- 6 Insure lying flat Save all urine specimen for toxicologic studies
- 7 Avoid dropping airway Aspirator on patient's tongue for  
ward and insure tracheal airway if tracheal intubation may be advisable
- 8 Oxygen hypoxia pressure and automatic yelling device  
(Page 150) may be valuable
- 9 Antibiotic drug Penicillin 300,000 to 500,000 units IM  
daily to prevent development of pneumonia
- 10 Potassium chloride If pulmonary edema is severe give  
1 liter of physiologic saline and 1.5 liter of 5% dextrose  
1/4 in water daily If pulmonary edema is present do not  
saline solution but give hypertonic saline solution very  
slowly 1/2 liter if edema has been severe do not  
give more than 2 to 3 liters of fluid during first 24 hours  
if the evidence of increased electrolyte imbalance
- 11 Central nervous system stimulation (analgesic or convulsant  
drug) The drug is not true barbiturate but a  
stimulant mainly in part to stimulate reflexes  
The drug is to be used in those who are  
symptomatic severe pallor or cyanosis or who  
condition is progressive They are dangerous and  
unless used with extreme care the risk of a chance  
for convulsions Superintendence of the physician is essential  
for over all observation and treatment
- a Potassium chloride in 1:500 (0.2% or 2 mg/cc)  
Administer 2-3 cc IV (or IM) at once as follows with  
1 very 20-30 minute until return of reflexes small  
increments as needed adjustment (not convulsions) if  
in 4-6 hours does not respond keep patient at a level  
b Potassium chloride 1:500 (2 mg/cc) 1:500 (2 mg/cc)  
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over the above treatment methods

- 12 Dialysis of the patient's blood with the artificial kidney is indicated in severe cases when available

### BELLADONNA DERIVATIVES

(Atropine code No 010-362) (Scopolamine code No 010 379)

The belladonna alkaloids are parasympathetic depressants with variable C M S effects M L D is 2.5 mg ( $\frac{1}{30}$   $\frac{1}{10}$  gr) of atropine sulfate but the usual lethal dose is nearer 100 mg ( $1\frac{1}{2}$  gr) The patient complains of dryness of mouth thirst difficulty in swallowing and blurring of vision The physical signs include dilated pupils flushed skin tachycardia fever delirium delusions paralysis and stupor

#### Treatment.

- 1 Tincture of iodine 4 cc (1 dr) in 1000 cc (1 qt) of water
- 2 Universal Antidote charcoal in water (See back cover)
- 3 Lavage well with 1:2000 potassium permanganate solution
- 4 Magnesium sulfate 30 Gm (1 oz) in water
- 5 Pentobarbital sodium 0.1 Gm ( $\frac{1}{12}$  gr) for excitement
- 6 Avoid opiates
- 7 Institute supportive measures

### BROMIDES

(Acute code No 010-3217) (Chronic code No 011 3217)

Bromides are C N S depressants frequently found in medicinal preparations Acute poisoning is rare The symptoms include anorexia constipation drowsiness apathy and hallucination The physical examination reveals dermatitis conjunctivitis foul breath furred tongue sordes unequal and irregular pupils ataxia abnormal reflexes (often bizarre) toxic psychosis delirium and coma

#### Treatment.

- 1 In acute poisoning, lavage copiously with saline to remove unabsorbed bromides and later to remove those excreted into stomach Follow with magnesium sulfate 30 Gm (1 oz) in water for catharsis
- 2 Sodium chloride 5-12 Gm (90-180 gr) daily in addition to regular dietary intake 1000 cc saline I V b i d or the same rectally or 1-2 Gm ( $\frac{1}{4}$ - $\frac{1}{2}$  gr) every 4 hours orally
- 3 Force fluids to 4000 cc daily
- 4 Use continuous warm baths (95-98 °F) or sedative cold packs as necessary
- 5 Otherwise treat symptomatically

### CARBON MONOXIDE (code No 010 352)

This gas is responsible for many deaths and near deaths resulting from the use of unvented gas or coal burning heaters It is also for suicidal purposes It combines with hemoglobin to form a

relatively stable compound which has caused laryngeal edema and  
 Manifestations are headache, faintness, giddiness, tinnitus, vomit-  
 ing, hyperaesthetic skin, rigors, loss of memory, fainting, collapse,  
 paralysis and unconsciousness as

Laboratory Data When boiled or when shaken with 1 to 2 volumes  
 of sodium hydroxide, blood remains red while normal blood  
 becomes black or brown black.

#### Treatment

1. Move patient to fresh air, keep warm, loose clothing  
 and mouth intact.
2. Inhalation of oxygen.
3. Artificial respiration or resuscitation as needed.
4. Give 50 cc of 50% glucose I.V. for cerebral edema.
5. Institute supportive measures.

### CARBON TETRACHLORIDE (code No. 010 33411)

This is the very common one in industry and the home be-  
 ing used as a solvent and cleaning agent. It is a local irritant  
 and causes an irritant contact dermatitis. It is a local irritant  
 to the mucous membranes which has a marked effect on the liver and  
 kidneys. It enters the body by ingestion and inhalation. The M.L.D.  
 is 4 cc (1 dr.) when ingested. The M.L.D. by inhalation is unknown.  
 The symptoms include headache, dizziness, nausea, vomiting, diar-  
 rhea, abdominal pain, decrease in visual disturbance, irritability  
 and intoxication. Early signs are tenderness in the epigastrium  
 and urinalysis shows epigastria and cirrhosis of the

#### Treatment

##### A. Acute Poisoning

1. Remove from exposure, keep patient warm.
2. Calum gel on the tongue 10 cc (2 1/2 dr.) of 10% solution I.V.  
 in 5 minutes.
3. Label copiously with 1:2000 potassium permanganate  
 solution.
4. Magnesium sulfate 30 Gm (1 oz.) in water at once.
5. Treat as potential acute hepatitis (see page 279). Observe  
 for oliguria. If it becomes manifest, treat as acute  
 failure (see page 303).
6. Institute supportive therapy.

##### B. Chronic Poisoning

1. Remove from exposure.
2. Carefully evaluate heart, liver and kidney function.
3. High protein, high CHO, low fat diet (see page 53).
4. Protein hydrolysis and glucose in line or water 2000  
 3000 (3 qt.) I.V. daily. Continue until fluid intake is taken  
 5. Calum gel on the tongue 10 cc (2 1/2 dr.) of 10% solution I.V.  
 bid and 12 Gm (2 3/4 dr.) by mouth bid.
6. Treat as potential cirrhosis (see page 280).
7. Symptomatic and supportive measures.
8. Avoid alcohol.

## CYANIDES (code No 010 353)

Hydrocyanic acid and the cyanides cause death by inactivation of the respiratory enzyme preventing utilization of oxygen by the tissues. The acid is most lethal and the M L D is 2 cc (1/2 dr) either by ingestion or inhalation. There is a rapid onset of giddiness, loss of muscle power and stupor accompanied by panting respiration and by profound collapse. The odor of bitter almonds is on the breath.

### Treatment

Work rapidly for death occurs quickly

#### A If inhaled

- 1 Place in open air keep in recumbent position
- 2 Maintain artificial respiration manually until arrival of resuscitator
- 3 Amyl nitrite (place pearls inside mask) by inhalation for 15-30 seconds every 2 minutes
- 4 Sodium nitrite 10-15 cc (2 1/2-4 dr) of 3% solution I V (or 50 cc of 1% solution) taking 2-4 minutes for injection

#### B If ingested lavage stomach copiously with 3% hydrogen peroxide solution 10% sodium thiosulfate solution or 0.2% potassium permanganate

#### C Supportive therapy

## DDT (Dichloro-diphenyl trichloro ethane) (code No 010 3 )

DDT is a CNS stimulant which can cause poisoning by ingestion, inhalation or direct contact. The M L D is probably about 20 Gm (5 dr) but few fatalities have been reported. When poisoning occurs from the material in solution the actual poisoning is usually due to the organic solvent and not DDT. The manifestations are tired and aching limbs, nervous irritability, mental sluggishness, muscle twitchings, convulsions and coma.

### Treatment

- 1 Universal Antidote at once if available (see back cover)
- 2 Lavage with large quantities of warm water
- 3 Magnesium sulfate 30 Gm (1 oz) in water
- 4 Phenobarbital sodium 0.1 Gm (1 1/2 gr) orally
- 5 Calcium gluconate 10 cc (2 1/2 dr) of 10% solution I V for convulsions
- 6 Avoid epinephrine. May cause ventricular fibrillation
- 7 Supportive measures as necessary
- 8 High CHO and high protein diet with vitamin B supplements to protect liver

## FLUORIDE POISONING (code No 010 3215)

Fluorides are found in agricultural poisons and insect powders and are used in the aluminum industry. Clinical features include vomiting, colicky abdominal pain, diarrhea, cyanosis, CNS excitement and convulsions.

- T atm nt
- 1 Lim w i r o ally in larg quantiti
  - 2 Gi e emetic or use copious gastric l vage with lime wate
  - 3 Egg whit b ted with 500 cc (1 pt ) milk or w i r
  - 4 Stim lant (se page 533)
  - 5 Calcium gl onat 10 c of 10% s lution I y rep at if tetany occurs
  - 6 A tificial r pir tion
  - 7 Combat shock

### GASOLINE AND RELATED COMPOUNDS (code No 010 332)

G s line poisoning may result ith r from Inhal tion ring s tion but m e acver symptom r suit from inhalation beca e th C N S i more quickly re hed by this route Th manif tations in the cute f rm e omiting v rtigo m cul r in co dination weak and ir regula pulse twt hing and onvulsions In the hro ic fo m th e is also h dache drow in e dim vision cold and n mb hand w akn lo of memory lo s of weight ta hy ca di m tal dcline e o confusion so e in mouth d m t es and s ndary an mia

- Tr tm t
- 1 R move to f ash al
  - 2 Lav ge with lad oil and/o larg amounts f warm saline
  - 3 M gnesium ulf t 30 (1 oz ) in wat followed by min al il 120 c (4 oz )
  - 4 W i h l s ly for 3 or 4 days for sev symptoms and fo collapse

### IODINE POISONING (code No 010 3216)

Clini l f stures in lade ha a teriestic tain of mouth and odo f b th yell w or bluish vomitu p in and b i g in ph ynx and ophagu m k d thirst diarrhea (stool m y be bl ody) we kn di stines yncope o onvulsions

- Tr atm t
- 1 Gi ta ch, flow r w gg whit o 1% sodi m thi l f t i wat by mouth
  - 2 P il w with m tic or r move by lavage with 1% sodium thi sulf te lution Repeat until eviden e f iodin ha di ppeared f om g at ic ont i
  - 3 The give dem lcenta e g milk or b i y wat r
  - 4 Symptom ti and supportive m u es for syst mic r tion f stimula t o anti onvul ants

### LEAD POISONING (code No 010 3112)

Le d m y po son by ing tion Inhal tion of its f m It has a local ast ing at action and a ge lli ed t ic efr ct The M L D is 10 Gm (150 gr ) f i d a tat P i oning is manif at d by

## 542 Poisons

metallic taste dry throat thirst abdominal colic vomiting diarrhea constipation headache leg cramps black stools (lead sulfide) oliguria stupor convulsions palsies and coma In the chronic form there is variable involvement of the C N S blood forming organs and gastrointestinal tract

### Treatment

#### A Acute Poisoning Do not use BAL®

- 1 Lavage with dilute magnesium sulfate or sodium sulfate solution to precipitate insoluble lead sulfate
- 2 Treat symptomatically Avoid narcotics treat colic with local heat antispasmodics and sedatives
- 3 Mono calcium disodium ethylenediamine tetra acetate (Calcium E D T A or Calcium Disodium Versenate Solution) Intravenous® This agent forms a soluble un ionizable lead complex that is excreted in the urine and has apparently been used successfully in the treatment of lead poisoning Dosage varies 300 600 cc (10 20 oz ) 5% solution over a 2 hour period I V daily for 5 10 days

#### B Chronic Poisoning

- 1 Remove permanently from exposure
- 2 Adequate diet with vitamin supplements
- 3 Courses of Calcium E D T A may be employed especially when hematological complications have occurred to rid the body of lead (see above)

## MERCURY (code No 010 3111)

Mercury poisoning occurs by ingestion or inhalation It is a general protoplasmic poison The M L D is about 70 mg (1+ gr ) of mercury bichloride The manifestations include metallic taste salivation thirst burning sensation in throat discoloration and edema of oral tissues abdominal pain vomiting bloody diarrhea and shock In the chronic form there is weakness ataxia intention tremor irritability depression and muscle cramps

### Treatment

- 1 Give white of eggs beaten with water or skimmed milk
- 2 BAL® must be started at once (see page 536)
- 3 Magnesium sulfate 30 Gm (1 oz ) in water
- 4 Fluids 1000 cc (1 qt ) of saline I V at once (may add 1 Gm sodium thiosulfate) and repeat as necessary
- 5 Watch urinary output Treat oliguria and anuria if it occurs (see page 303)
- 6 Symptomatic and supportive measures as necessary
- 7 In chronic form remove from exposure may give 10 Gm (15 gr ) of sodium thiosulfate in 100 cc (2 1/2 dr ) water I V every other day

## MORPHINE (AND THE OPIATES) (code No 010 370)

Morphine acts primarily on the C N S causing depression and The M L D is 65 mg (1 gr ) in susceptible individuals

Mife tations incl de be da h n us ex item nt d p e si n  
 in point p pil slow respirations r pid nd f ebl puls ho k  
 and oma

T m t

- 1 N lorphin Hyd ochlorid N N R (N lline®) a narcoti  
 tagonist in dos a of 5 10 mg I V as antidote for  
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 mg m y h epe t d in 10 to 15 minut s and again d pending  
 upon th degr and d at n f na oti dep essi n
- 2 M ltain adequal re pirat on by u of artifl al re pirat on  
 pref bly aus tat s with o yge
- 3 Ke p p tient awak and w rm have him w lk if nec ssa y or  
 use mmo la inhalat on a d t ong stunult
- 4 Antidot of 4 c (1 dr ) tin tur of Iodin in quart of wate  
 if taken ally
- 5 L vag tomach well with 1 2000 potassium permanganat at  
 short interval M rphin is e eted into the stom ch
- 6 Magn lum lf te 30 Gm (1 o ) in w te as catha ti
- 7 Atropi sulfat 0 5 mg (1/20 gr ) subc t if re pirati s  
 a e poo pe t as ne sary

MUSHROOMS (code No 010 384)

P i oning du to th Amanita ph illoid and th Amanit mus  
 caria i manife t d by se coli thir t naus and vomiting  
 p of diar e a, w akne collap heavy bre thing slow p l e  
 con t i t d pupil onfu lion excit m nt oliguri and coma

T m t

- 1 Atropin e lfai 0 5 1 0 mg (1/20 1/80 gr ) sbcut at once  
 and pe t as needed. (Antidot fo mu rini a tion )
- 2 Uni e al Antidot whit of ggs in milk or 1 tsp  
 tin tur of Iodin in a quart of w ter
- 3 Lavag with 1 2000 potas lum pe manga at olutio
- 4 Magne lum ulf t 30 Gm (1 o z ) in w ter a c thartic
- 5 P tobarbital odiam, 0 2 Gm (3 gr ) orally fo e item t
- 6 Bismuth a b bo ste 1 0 Gm (15 gr ) ev y 2 hours fo  
 g t itis
- 7 High s line e m to help pre t ab orption
- 8 Fo fluids by mouth and par nt al routes
- 9 Institut supportive meas Wait h fo hepa

OXALIC ACID (code No 010

Oxalic acid a component of bl achin  
 local ir itant whi h p cipitat a ionized  
 4 Gm (1 dr ) Poisoning is manife ted b  
 throat viol t abdominal pain bloody vo  
 oliguria, and circulatory c llap



## SNAKE (AND GILA MONSTER) BITES (code No 010 3814)

The venom of poisonous snakes and lizards may be neurotoxic or hemotoxic. Neurotoxin causes respiratory paralysis, hemotoxin causes hemolysis and destruction of endothelial lining of blood vessels. The manifestations of poisoning are local pain, thirst, profuse perspiration, nausea, vomiting, stimulation followed by depression, local redness, swelling, extravasation of blood and collapse.

### Treatment

- 1 Keep patient recumbent and quiet
- 2 Apply tourniquet above bite releasing for 1-2 minutes every 15 or 20 minutes
- 3 Cut deep cross incisions at site and apply suction
- 4 Give specific antivenom (Follow printed instructions)
- 5 Plenty of warm fluids, no alcohol (synergistic with venom)
- 6 Barbiturates if sedation is needed. Avoid opiates
- 7 Institute supportive measures, transfusions if necessary

## SPIDER BITES (code No 010 3815) AND SCORPION STINGS (code No 010 3815) (Black Widow Spider Bite code No 010-3816)

The toxin of the less venomous species of spiders and scorpions causes only local pain, redness and swelling. That of the more venomous species causes generalized muscular pains, convulsions, nausea, vomiting, variable C I S involvement and collapse.

### Treatment

- 1 Apply tourniquet, cut cross incisions and apply suction
- 2 If absorption has occurred give 10 cc of 10% calcium gluconate I V or I M. Repeat as necessary
- 3 Give specific antivenom (Follow printed instructions)
- 4 Keep patient recumbent and quiet
- 5 Hot baths and 20 cc of 10% magnesium sulfate I V for relief of pain (see caution on page 296)
- 6 Adequate sedation. Institute supportive measures
- 7 Hot compresses of sodium bicarbonate solution for relief of local pain if no systemic involvement
- 8 ACTH or adrenal steroids may be of some value (see page 423)

# APPENDIX

## MEDICAL REHABILITATION

By rehabilitation medical rehabilitation or rehabilitation of the physically handicapped patient is meant the restoration of the patient to his maximum physical, social and economic capacity. It differs from medical rehabilitation in that the aim of the latter is to cure illness and to prolong life.

Rehabilitation has also been called the third phase of medical care. The three phases are (1) prevention of illness, (2) definitive medical care, and (3) rehabilitation.

Rehabilitation is not and should not be delayed until definitive medical care has been completed to be most effective it should start at the onset of any illness acute or chronic. Frequently definitive therapy and rehabilitation are difficult to reconcile. Proper late compromises must be made in order to render the best service to the patient.

Many a residual disability is not caused by the original illness but by inactivity (eg bed rest or hospitalization) or faulty positioning. Both of these can be prevented. Flexion contracture of the elbow is a painful condition in hemiplegia and my ardi in far from easily visible if rehabilitation starts early. Furthermore the psychologist's outlook for the patient (and thus his cooperation with therapy) improves when he knows the goal is not only cessation of illness but restoration to usefulness as well.

## REHABILITATION OF THE HEMIPLEGIC PATIENT

Advances in physical medicine have given new hope to the patient who suffers from hemiplegia a condition which is counted down and moribund in the medical profession. The following program is intended to serve only as a guide it applies to the typical case of cerebral vascular accident but the principles are the same in hemiplegia of any etiology.

### Bed Phase

Start on second or third day of illness or as soon as the patient is conscious. The patient should be of fair height and should have idiosyncrasies and an overhanging trapeze.

**A Exercises** Start with ten minutes of exercise every two hours and increase to 15 minutes of exercise every two hours.

- 1 With good arm and leg, turn from back to side to abdomen, then to other side and then back. Repeat in opposite direction.
- 2 With good hand on trapeze pull to sitting position and back.
- 3 Move sideways up and down in bed.
- 4 Sit up on edge of bed with side rail moved, legs dangling and move along edge of bed with aid of good arm and leg.

**B Self Care** (all done with good hand)

- 1 Toilet activities Wash face and hands comb hair shave.
- 2 Feeding activities At first in bed with back rolled up later sitting on edge of bed.

**C Bedding** None during bed phase.

Standing Phase

Starts three to five days after beginning bed phase and should replace bed phase as soon as possible. Patient is placed in an arm chair with his good side next to the bed. The vertical bar of the overhead frame is reached by his good hand and the paralyzed arm is in a sling.

**A Exercise** Start with ten minutes of exercise every two hours and increase to 30 minutes every two hours.

- 1 With good arm pull to standing position on good leg. Sit back.
- 2 Standing with good hand on vertical bar of overhead frame perform slight knee bend and straighten up. Repeat with gradually deeper knee bends.
- 3 Stand with good hand on vertical bar of bed frame. Go up on toes, come back down.

**B Self Care (using good hand)**

- 1 Toilet activities. Complete bath in bed.
- 2 Dressing activities. Dress and undress except for shoes.

**C Bracing**

- 1 Flat wooden splint (attached to volar surface with ace bandage or straps) from one inch below the elbow to one half inch beyond the fingertips of the paralyzed arm.
- 2 Keep paralyzed arm in sling to prevent pull on shoulder.
- 3 If after two weeks the paralyzed leg still swings completely flail at the knee joint, a long leg brace is needed in order to continue rehabilitation.

Stair climbing Phase

Starts two to ten days after the beginning of the standing phase and should replace standing phase as soon as possible.

**A Exercises** Performed four times a day increasing from several steps to a whole flight of stairs. The patient is placed in a chair facing the foot of a flight of stairs. The good arm next to the banister. The paralyzed arm is splinted and in a sling and the paralyzed leg is in a long leg brace if needed.

- 1 Pull to standing position holding to the banister with the good hand. Step up one step with the good leg, then pull paralyzed leg up to the same step. Continue for several steps.
- 2 Step backward and down with the paralyzed leg and put the good leg down next to it. Continue for several steps.
- 3 While several steps up turn towards and reach over to the opposite banister. Step forward and down with the paralyzed leg. Then place good leg next to paralyzed leg and continue.

**B Self Care** Complete toilet and feeding and dressing activities should be possible by this time.

**C Bracing**

- 1 Long leg brace if indicated.
- 2 If patient has a foot drop during stair climbing, he should have a short leg brace with a 90° posterior stop at ankle.
- 3 If the patient shows evidence of inversion or eversion of the foot, he should have a short leg brace with a T strap.
- 4 If function has returned to the paralyzed hand, the splint may be discarded. Otherwise it should be worn intermittently.

Walking Phase

Start as soon as the patient is capable of walking up and down a

whole flight of stairs with sitting Paralyzed arm is kept in sling  
and one is held with good hand Two different slings can  
be recommended for the hemiplegic patient  
A W G It (For feeble patients or patient with poor balance)  
Place one forward place good foot next to can and then drag  
paralyzed foot next to good foot

B F at G It  
1 Stand on good leg place one and paralyzed leg forward  
simultaneously and put weight on them  
2 Swing good leg through front of cane and paralyzed leg and  
put weight on it Continue in this fashion

Special Problem in Hemiplegic Patient  
C Th Paralyzed Upper Extremity  
1 Complete loss of function in most cases no useful func-  
tion remains to the paralyzed upper extremity and the wrist  
and hand are supported in the splint The sling may be  
drawn distal to the wrist should muscles be strong pasti-  
and the patient is limited by the sling With his good hand  
the patient should move the paralyzed finger wrist and  
thumb through the full range of motion twice a day in ord-  
er to move the paralyzed shoulder through the full range of  
motion the patient may do this through an overhead pul-  
ley by means of which the paralyzed arm (tied to the wrist)  
can be pulled up as high as possible with the good arm  
2 Partial function If only partial action remains to the para-  
lyzed extremity the patient should use only to the extent  
to which it is helpful expedient For the activities the  
patient should be trained in the use of the good extremity  
3 Complete function If complete function returns the patient  
should use the extremity as much as possible

B T  
In the hospital should be started as soon as possible If  
a neurologic aphasia is present the above program  
may be deferred until it is easier to understand of him  
ability of the patient to understand what is required of him

C C of Hemiparesis  
In the patient who would be trained in his hand to the  
hemiparesis aid in order to bring his visual field in front of  
him Later a more adjustment in the visual field occurs  
C of Spinal Cord Some hemiplegics experience incontinence  
lymph An indwelling catheter rarely necessary Th  
patient should be reminded to empty his bladder voluntarily at  
hourly intervals The intervals can be gradually increased

E Organic Mental Syndrome When this is present the whole  
habilitation program may be difficult The patient may with-  
stand treatment at one time and absolutely another and advantage  
should be taken of the patient's individuality Organic mental  
syndrome occurs usually in patients who have had severe  
stroke The patient's mental state usually improves or  
gradually regains a more habituation program outside

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## NORMAL HEMATOLOGICAL VALUES

White Blood Cells 5 000 10 000 per cu mm			
Myelocytes	0 %	Lymphocytes	40 %
Juvenile Neutrophils	0 %	Eosinophils	1 3 %
Band Neutrophils	0 5 %	Basophils	0 1 %
Segmented Neutrophils	40 60 %	Monocytes	4 8 %
Platelets 200 000 to 500 000 per cu mm			
Red Blood Cells in million per cu mm			
Men	5 0 (4 5 to 6 0)	Women	4 5 (4 3 to 5 5)
Reticulocytes Less than 1 %			
Hemoglobin in Gm /100 cc			
Men	15 (13 5 to 18)	Women	13 5 (12 5 to 15 5)
Hematocrit (packed cell volume)			
Men	45 47 % (38 54 %)	Women	40 42 % (36 47 %)
Cellular Measurements of r b c			
Average diam	7 3 $\mu$ (5 5 to 8 5 $\mu$ )		
Mean Corpuscular Volume	87 c $\mu$ (80 94 c $\mu$ )		
Mean Corpuscular Hb	30 $\gamma$ (28 3 $\gamma$ )		
Mean Corpuscular Hb Conc	35 % (33 38 %)		
Color Saturation and Volume Indices each 1 0 (0 9 1 1)			
Bleeding Time Dak 1 4 minutes Ivy less than 4 minutes			
Coagulation Time Lee and Whit 5 15 minutes			

## NORMAL BLOOD CHEMISTRY VALUES

	Constituent	Value/100 cc	mEq /Liter
Serum or Plasma	Sodium	310 340 mg	135 145
	Chloride (as Cl)	350 375 mg	100 106
	Total Chlorides (as NaCl)	580 6 0 mg	100 108
	Potassium	14 20 mg	3 5 5 0
	Phosphorus	3 4 5 mg	0 8 1 5 (mM)
	Magnesium	1 3 mg	1 2
	Calcium total	9 11 mg	4 5 5 5
	CO <sub>2</sub> Combining Power	55 75 Vol %	24 28
	Cholesterol	150 240 mg	
	Cholesterol esters	65 % of the total Cholesterol	
	Amylase	80 180 Units	
	Phosphatase alkaline	2 0 4 5 Units (Bodan ky)	
	Phosphatase acid	0 5 2 Units (Bodansky)	
	Protein Binding Iodine	4 8 micrograms	
Blood	Serum Albumin	4 5 5 5 Gm	Total
	Serum Globulin	1 5 3 0 Gm	6 0 8 0 Gm
	Fibrinogen (plasma)	0 2 0 6 Gm	per 100 cc
	Glucose 50 100 mg (true)	80 120 mg (Folin Wu)	
	Total Non protein Nitrogen	15 35	
	Urea Nitrogen	10 20	
	Uric Acid	3 6	
	Creatinine	1 2	

## NORMAL RENAL FUNCTION AND URINE VALUES

Phenol Red Test (P S P) 15 minutes over 25% 2 hours over 55%	
Urea Clearance 75 120% of A N F or 40 100 /minute	
Addis Urine Sediment Count (Values for 12 hour period)	
pH acid	Sp Gr 1 025 1 030
Albumin 0 30 mg	
b c 0 1 000 000	Calculation 100 000
w b c and small epithelial cells 0 2 000 000	

# ABBREVIATIONS USED IN THIS HANDBOOK

ā	Of e h	mEq	Milli q ival t
a	B fore m ls	mg	Milligram
ad lib	At ple su	μ	Minim*
amp	Amp l *	mm	Millim t r
b i d	Twice a d y	M L D	Minim m l th i doo
b m	Bow l mo em nt	γ	M crog am
B M R	Bas l m t bolic te	μγ	Mi o microgram*
B P	Br tish Pharmacopoei	μ	Mi ron
B U N	Blood ur a nit g l	N C A	Not Coun l Accepted
c	C p	N N R	N wand No off c al R medi s
C l	C lorie	N P N	Nomp t i nit ogen
ps	C psule	N R C	Nation l Resear h Coun il
C B III	Complel blood count: RBC WBC Diff Hgb	os or	Oun
cc	C bic ce tim t	P A	Perni io s an mia
cf	Conf	p c	After m als
CHO	Ca bohyd t	ppt	P e pit t d
C l	Col inde	p n	A s y
m	Centimete	P S P	Ph ois lph phthal in ph l d
C N S	C tral nervou syst m	Pe	Physi l gi al l e l cion
C S F	C b ospinal fluid	pt or O P nt	
u	Cubic	q	E ery
μ	Cubi mic on	q i d	F r times d y
dr o	D am	q s ad	S ff i e t t m ke up to
E g EKC	El t oca diogram	qt	Qu t
GB	G libladd	r b c	Red blood corp l
GI	G st oint atinal	RBC	R d blood cou t
Gm	Gram	R	P ript on
gr	G ain	c	2 ond
fl	Drop (gtt d op )	S i	S t tion inde
H	H	S g	Let it be label d
Hgb	H moglobin	Sol	o lul on
h s	At bedtim	p gr	pe ific gravity
I M	Int am c la ty		On half
I U	Int rn tion i unit	St t	Imm di tely
I V	Int no ly	s b ut	Subcutan ou ly
Kg	Kilogram	tab	Tablet
L	Lit r	Tb p	Tabl poon
lb	Pou d	t i d	Thr tim s a day
liq	Liq fl	t p	T a poon
mc	Millicurie	U	U it
M C H	M an corpus lar b moglobin	U S P	U S Pharmacopoi
M C H C	M an co pu cul b moglobin once t ation	V i	Vol m i d
M C V	M an rpuscular volum	w b	Whit blood c ll
		WBC	Whit blood count
		wt	W ight
		>	Gr t r than
		<	Le tha

Abb eviations for either singular o pl al



# TABLES OF APPROXIMATE EQUIVALENTS

Weight Equivalents		Volume Equivalents	
Apothecary	Metric	Apothecary	Metric
1/30 gr	0.2 mg	1 min (γ)	0.06 cc
1/210 gr	0.3 mg	3 min (γ)	0.18 cc
1/150 gr	0.4 mg	5 min (γ)	0.3 cc
1/100 gr	0.5 mg	8 min (γ)	0.5 cc
1/100 gr	0.6 mg	10 min (γ)	0.6 cc
1/80 gr	1.0 mg	12 min (γ)	0.75 cc
1/30 gr	2.0 mg	15 min (γ)	0.9 cc
1/16 gr	4.0 mg	18 min (γ)	1.0 cc
1/12 gr	5.4 mg	20 min (γ)	1.2 cc
1/10 gr	6.5 mg	30 min (γ)	1.8 cc
1/8 gr	8.0 mg	50 min (γ)	3.0 cc
1/6 gr	11.0 mg	1 fl dr (γ)	3.7 cc
1/4 gr	18.0 mg	65 min (γ)	4.0 cc
1/3 gr	22.0 mg	80 min (γ)	5.0 cc
3/8 gr	24.0 mg	2 fl dr (γ)	7.5 cc
1/2 gr	3.0 mg	2 2/3 fl dr (γ)	10.0 cc
3/4 gr	50.0 mg	4 fl dr (γ)	15.0 cc
1 gr	65.0 mg	5 1/2 fl dr (γ)	20.0 cc
1 1/2 gr	0.1 Gm	8 fl dr (γ)	1.0 fl oz
2 gr	0.13 Gm	1 fl oz (γ)	30.0 cc
3 gr	0.2 Gm	1 2/3 fl oz (γ)	50.0 cc
5 gr	0.32 Gm	2 fl oz (γ)	60.0 cc
7 1/2 gr	0.5 Gm	3 3/8 fl oz (γ)	100.0 cc
10 gr	0.65 Gm	4 fl oz (γ)	1.0 fl oz
15 gr	1.0 Gm	8 fl oz (γ)	240.0 cc
1 dr (3)	4.0 Gm	16 fl oz (γ)	480.0 cc
1 oz (γ)	30.0 Gm	1 pt	480.0 cc

Household Measures	Apothecary	Metric
1 teaspoon	1 fl dr (3)	4 cc
1 tablespoon	1/2 fl oz (3)	15 cc
1 teacup	4 fl oz (3)	120 cc
1 glass (tumbler)	8 fl oz (γ)	240 cc
1 measuring cup	8 fl oz (γ)	240 cc
1 pint	16 fl oz (3)	480 cc

## CENTIGRADE TO FAHRENHEIT TEMPERATURES

C	F	C	F	C	F
35	95	37.5	99.5	40	104
35.5	95.9	38	100.4	40.5	104.9
36	96.8	38.5	101.3	41	105.8
36.5	97.7	39	102.2	42	107.6
37	98.6	39.5	103.1	43	109.4

## METRIC SYSTEM

Weight	1 000 micrograms (γ)	1 milligram (mg)
	1 000 milligrams (mg)	1 gram (Gm)
	1 000 grams (Gm)	1 kilogram (Kg)
Volume	1 000 cubic millimeters	1 milliliter (ml)
		or 1 cubic centimeter (cc)
	1 000 cubic centimeters (cc)	1 liter (L)

# IDEAL WEIGHT FOR ADULTS AGES OF 25 AND OVER

(Country: British Metropolitan Life Insurance Company)

Height (With Shoe)		Ideal Weight in Pounds and Kilograms for MEN (For weight without shoe or clothing, but 15.5 pounds)					
		Smaller		Medium		Larger	
Feet	Cms	Lb	Kg	Lb	Kg	Lb	Kg
5 2	157.5	114 125	51.8 56.7	124 133	56.3 60.8	131 142	59.4 64.4
5 3	160.0	119 128	54.0 58.1	129 136	57.6 61.7	135 144	60.3 65.3
5 4	162.6	123 132	55.3 59.9	130 140	58.9 63.5	137 149	62.1 67.6
5 5	165.1	126 136	57.1 61.7	134 144	60.8 65.3	141 153	63.9 69.4
5 6	167.6	129 135	58.5 62.1	137 147	62.2 66.7	145 157	65.8 71.2
5 7	170.2	133 143	60.3 64.9	141 151	64.0 68.5	149 160	67.6 73.5
5 8	172.7	136 143	61.7 66.7	145 156	65.8 70.8	153 166	69.4 75.3
5 9	175.3	140 151	63.5 68.5	149 160	67.6 72.6	157 170	71.2 77.1
5 10	177.8	144 155	65.3 70.3	153 164	69.4 74.4	161 175	73.0 78.4
5 11	180.3	148 159	67.1 72.1	157 168	71.2 76.3	165 180	74.8 81.7
6 0	182.9	152 164	69.0 74.4	161 173	73.0 78.5	169 185	76.7 83.9
6 1	185.4	157 169	71.2 76.7	166 178	75.3 80.7	174 190	78.9 85.2
6 2	188.0	162 175	73.9 79.4	171 184	77.6 83.5	179 195	81.2 88.9
6 3	190.5	166 180	76.2 81.7	176 189	79.8 85.7	184 202	83.5 91.8

Height (With Shoe)		Ideal Weight in Pounds and Kilograms for WOMEN (For weight without shoe or clothing, but 12.3 pounds)					
		Smaller		Medium		Larger	
Feet	Cms	Lb	Kg	Lb	Kg	Lb	Kg
5 0	152.4	107 113	47.6 51.3	112 121	50.8 54.6	119 129	54.0 58.5
5 1	154.9	107 115	48.5 52.2	114 122	51.7 55.3	121 131	54.9 59.4
5 2	157.5	110 118	49.9 53.5	117 125	53.1 56.7	124 135	56.3 61.2
5 3	160.0	113 121	51.2 54.9	120 128	54.4 58.1	127 138	57.8 62.6
5 4	162.6	116 125	52.8 56.7	124 132	56.3 59.9	131 142	59.4 64.4
5 5	165.1	119 128	54.0 58.1	127 135	57.6 61.7	135 146	60.3 65.3
5 6	167.6	123 131	55.8 60.9	130 140	58.9 63.5	138 150	62.1 67.6
5 7	170.2	126 135	57.2 61.7	134 144	60.8 65.3	142 154	64.4 69.4
5 8	172.7	129 139	58.5 63.1	137 147	62.2 66.7	145 158	65.8 71.2
5 9	175.3	133 143	60.3 64.9	141 151	64.0 68.5	149 162	67.6 73.5
5 10	177.8	136 147	61.7 66.7	145 155	65.8 70.8	152 166	69.4 75.3
5 11	180.3	139 150	63.1 68.5	148 158	67.1 72.1	155 169	70.3 76.7
6 0	182.9	141 153	64.0 69.4	151 163	68.5 73.9	160 174	72.6 78.9

Feet 14.1 25 pp m id i w gh an be 1 i d by  
bt cti g i 0 lb (0.5 Kg) f hy ar of g i han 25 y

## AVERAGE HEIGHT AND WEIGHT FOR CHILDREN

Age	BOYS				GIRLS			
	Height		Weight		Height		Weight	
	Feet	Cms	Lb	Kg	Feet	Cms	Lb	Kg
1 1/2	1 8	45.7	14.5	6.6	1 8	50.8	14.5	6.6
1	2 3	66.0	17	7.7	2 2	66.0	16	7.3
1	2 5	73.6	23	10.5	2 5	73.6	20	9.1
2	2 8	82.8	26	11.8	2 9	83.8	25	11.3
3	3 0	91.4	3	14.0	3 0	91.4	30	13.6
4	3 3	99.0	34	15.4	3 3	99.0	33	15.0
5	3 6	106.6	39	17.7	3 5	104.1	38	17.2
6	3 9	114.2	46	20.9	3 8	111.7	45	20.4
7	4 1	121.7	5	23.1	4 1	121.7	4	22.2
8	4 3	127.0	57	25.9	4 3	127.0	54	24.4
9	4 6	132.0	63	28.6	4 6	132.0	63	28.1
10	4 8	137.5	6	31.2	4 8	137.5	6	29.2
11	4 8	142.2	73	34.8	4 8	142.2	77	34.9
12	4 10	147.3	83	37.7	4 10	147.3	86	39.0
13	5 0	152.4	9	41.7	5 0	152.4	107	48.5
14	5 2	157.5	107	48.5	5 2	157.5	107	48.5
15	5 4	162.6	116	52.6	5 4	162.6	115	51.7
16	5 6	167.6	128	58.0	5 6	167.6	118	53.5
17+	5 7	170.2	134	60.8	5 7	170.2	119	53.5

\* Height g 13 and 17 stream by lah

# EMERGENCY DRUGS

Drug	P P tions and Do ges	A tion	I di tion	Contr t di tion and Cs tions
Anob bit 1 Sodium N Y (Amyt 1 Sod um)	0.25 0.50 Gm. powd. in c p sul a m k up as f sh 10% Sol. Give slowly I V 1 M 0.13 Gm (2 gr) rectal up 11 s	Hyp ot a dative antie val ant	Co vulsions t s s p l p t i c hy t - la, manu re t on to loc i are th ti	It bit t s nstlly deli sum r pirst ry distr a ( tr t) he p ti in utt i n y (give apid ti g bit at ) r t i i f f i n e y (give l w ti g b bit r t )
Par id hyd U S P B P	Or ly 4.16 c (1 4 d ) i d 2.3 lum (2.5 dr) with min al o ) 1 M 4.10 cc (1 2 1/2 dr) f v 2.3 cc diluted with t ip) v i m of ph i l g l i l i n	Hyp t c s d t i v e ti convulsant	Convul sion t t p l p xell roe t hy t r i mant re tion to loc i an th ti m y 2 be s d in t t m t of d i i m	It p tory di (p lmon y) h p t i t u f f i t y gastro- i t t i e l a l o t e )
M rphue Sul t U S P B P	8.32 mg (1/2 gr) sub t 1 M I V (For th lat) r ce line s tions)	N r t i l s d (an lg t s d hyp otic)	S w p u a d t m l i p l u s a pulmon yed m ver di the	H d injury m phi ti y bronchi l a s t m undiagn d s r g l al below in l d i s e h e p t i t f f i t y h y p o t h y r i d m
At opin S U t U S P B P	0.5 0.8 mg (1/2 0 1/100 gr) lly r be t i n e o u s l y	Par ymp ch n dep s a t C N S stim lant	C i hyp n m n l y l pol oning m rph n poisoning m h e o m poisoning roid l s y m o p e Stok Ad m s m o p e	Gl m drug n t i t y
Epun phrin U S P Ad line B P	0.1 0.5 (2 s w) of 1 1 000 1 t i o n be t 1 M I V	Symp th m m t	V i l l t a b d t i l l b o n h i c h m a n g l o n e u r o l d t m h o c k ( ? )	Ad an d h y p e i l v e v a l r d i s a n d c e r e b r a l r i l o r s i c o l a r i o u r e g a n i c h e r t d i d u r i n g r i a n t h i s
Amioophyllo U S P Th ophyllin with Ethylene di mine B P	0.25 0.50 Gm (2 3 4 7 1/2 g) 2.5% lution slowly I V 1 M or 0.50 Gm t i p p l o r y	B o n h i l d i t t m y o c a d i l t i o n l i t o o a y c o d i l a t ( ? ) d i a t i c	P l m o n y d m b o n h a l t h m a C h y n Stok s p l r t i o n m y o c d i l i n f t i o n ( ? ) t t g l n s ( ? )	



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